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THE UNIVERSITY OF ALBERTA

THE USE OF PLANT BREEDING AND CHEMICAL METHODS FOR THE CONTROL OF ALFALFA SICKNESS

by

© TYRONE R. FAECHNER

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled THE USE OF PLANT BREEDING AND CHEMICAL METHODS FOR THE CONTROL OF ALFALFA SICKNESS submitted by TYRONE FAECHNER in partial fulfilment of the requirements for the degree of Master of Science in Plant Breeding.

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ABSTRACT

Heat treatment, three fungicides (Dexon, Metazoloxon, and Dowco 269), and plant breeding methods were compared for effectiveness in the control of a pathogen that attacks the roots of alfalfa (_Medicago media_ Pers.). Of eleven growth room tests, eight were used to investigate physical and chemical methods of control and three to evaluate plants selected for resistance and susceptibility to alfalfa sickness. The mean height and disease rating per pot was determined as well as the total yield of plant dry matter per pot. Five field tests were conducted on an experimental area at Spruce Grove, Alberta where height, performance rating, and yield were recorded on the basis of single row plots. Disease rating was recorded as a plot mean.

Alfalfa sickness is caused by a pythiaceous fungus which will infect alfalfa seedlings at the root tips, lateral noncambial roots, and nodules. Browning and lesioning of the root tissue results leading to restricted growth and to shorter, lower yielding plants.

Pasteurization of sick soil provided an effective means of controlling alfalfa sickness in the growth room. Height was consistently increased while disease rating and yield were improved.

Fertility level, pH, and soil moisture content were shown not to be casuative agents of alfalfa sickness.

Dexon did not control alfalfa sickness, however

Metazoloxon and Dowco 269 did. Dowco 269 was much more effective than Metazoloxon, and may have a place in the commercial control of alfalfa sickness.

Alfalfa plants were selected for 2 cycles from within locally adapted cultivars and compared in two diallel crossing systems. Genotypic variability for resistance and susceptibility to the disease was evident from the high broad sense heritabilities. Low narrow sense heritability estimates suggested consecutive cycles of recurrent selection with well-replicated progeny tests would be necessary to breed high levels of resistance to the disease.

Selection progress was slow indicating that many genes may control the inheritance of resistance and susceptibility to alfalfa sickness. Recurrent phenotypic selection in the growth room was effective since increased levels of resistance were obtained and no change, in agronomic characters for which selection was not made, was observed.

Genetic variation was mostly due to nonadditive gene action in the diallel cross which tested plants selected for susceptibility since the specific combining ability (SCA) variance was larger than the general combining ability (GCA) variance. In the diallel cross which compared resistant genotypes, genetic variability was due to additive gene action since the GCA variance was larger than the SCA variance.

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INTRODUCTION

Alfalfa (Medicago sativa L. and Medicago falcata L. and hybrids between these species) is the most important perennial forage legume grown in western Canada. It thrives on nearly neutral soils, responds to irrigation, and will yield upwards of 4.5 metric tons of dry matter per ha. This legume requires well drained soil which it improves through its ability to fix atmospheric nitrogen. Alfalfa is a palatable, nutritious fodder with a crude protein content of approximately 17%.

Most of the cultivars of alfalfa originate from M. sativa and M. falcata . The falcata derived cultivars are considered to have more winterhardiness and drought resistance but lower seed and forage yields compared to cultivars originating from the sativa species. Canadian cultivars are the result of interspecific crosses between M. sativa and M. falcata and are usually known as Medicago media Pers..

Alfalfa is essential to the dairy, beef, and alfalfa dehydration industries. Census data (Alberta Agriculture, 1977) indicated there are 740,000 ha (or 6% of the cultivated area) in pure alfalfa stands and mixtures containing alfalfa in Alberta. The potential for expansion has been conservatively estimated at 1.5 million ha, more than double that now in production. The annual requirements of alfalfa for the dairy industry approached 225,000 metric tons for 1975 (Alberta Agriculture, 1975). Within the vincity of Edmonton there are six alfalfa dehydration plants producing about 12,600 metric tons, yearly, from an area of 3,200 ha per factory.

In central Alberta, a condition restricting the growth of alfalfa has been observed (Webster et al., 1967). This syndrome has been referred to as "alfalfa sickness" and the soil inciting it as "sick soil". In the disseration that follows, those terms will be used.

Poor growth of alfalfa in the Edmonton, Alberta area was first recorded by Goettel (1962) who found fertilizers did not correct the problem. Although the causative agent responsible for alfalfa sickness was not isolated, Webster et al (1967) indicated it was biological. A selection study which was undertaken by Goplen and Webster (1969) failed to demonstrate inheritance of resistance to alfalfa sickness. A nitrogen imbalance and possibly an aluminum toxicity were concluded to be the factors affecting alfalfa in sick soil (Webster and Dekock, 1970). Root zone temperatures in sick soil did not shed light on any new area nor did corrective fertilizer applications (McElgunn and Heinrichs, 1970). Webster et al. (1972) attempted, but did not succeed, in establishing a relationship between a species of nematode and alfalfa sickness. Subsquently, Damirgi et al. (1976) proved alfalfa sickness was not caused by the nematode. These authors produced symptoms typical of alfalfa sickness using sick soil serial dilutions.

The characteristic symptoms of alfalfa sickness have been described by Goettel, 1962; Webster et al., 1967; Webster and Dekock, 1970, and Damirgi et al., 1976, and may be recapitulated as follows.

1) The plants are stunted and chlorotic.

2) Irregular, brownish lesions appear on lateral and tap roots followed by girdling of the lateral roots.

3) Older roots do not exhibit deep necrotic areas but poor nodulation is evident.

Although the seriousness of alfalfa sickness has not been assessed in terms of yield loss, it has been observed (Bolton, 1977) that alfalfa stands on sick soil produce low yields. The disease may be controlled by studying the way in which it interacts with the plant, and by examining the response of the disease pathogen to chemical and physical treatments. However, such treatments are frequently expensive and thus of little practical value. Consequently, an attempt was made to obtain resistant progenies by plant breeding methods. Field observations revealed differences in the degree of field resistance within, as well as between, local cultivars. If these differences are inherited, then resistant plants could be selected. An attempt was also made to eludicate the genetic mechanisms which govern the expression of this trait. The object of the present study was, then, to control the alfalfa sickness pathogen by chemical and physical treatments and to identify resistant clones which can be used to synthesize a resistant cultivar.

LITERATURE REVIEW

1. Pathogen

a. Controversy From studies conducted at the University of Alberta, the casual agent of alfalfa sickness is a pythiaceous fungus (Cock, 1977; Damirgi *et al.*, 1977), the identity of which is controversial. Disagreement revolves around the classification of the pathogen as a *Pythium* sp. or a *Phytophthora* sp.. This disagreement results from three factors. First, the classification of the Phycomycetes is somewhat ambiguous (Walker, 1969; Waterhouse, 1973). Table 1 compares the morphological characteristics distinguishing the species and genus in question. Waterhouse (1970) admits that *Phytophthora megasperma* Drechs. is a difficult species to classify and there is a question of speciation with the *megasperma* complex.

Second, the primary invader is confused with secondary invaders. Erwin (1954b) suggested secondary organisms might suppress or mask *Phytophthora cryptogea* Pethybridge and Lafftery in alfalfa roots during unfavorable conditions for this pathogen. Frosheiser (1968) stated *P. megasperma* was endemic to the soil only requiring suitable environmental conditions to develop. This is further supported by the widespread geographic distribution of *P. megasperma* reported in the literature. Bearing in mind these observations plus the fact that no *Pythium* sp. have been identified as the casuative agent of a roct rot of alfalfa in western Canada, the initial invader causing alfalfa sickness was believed to be a *Phytophthora* sp..

The third factor was time for resistance to develop in alfalfa against *Pythium* sp. as compared to *Phytophthora* sp.. Haplin and Hanson (1958) found alfalfa to be susceptible to 5 species of *Pythium* but immunity to *Pythium* sp. was developed 3 days after seeding. Chi and Hanson (1962) reported similar results for resistance to *Pythium debaryanum* Hesse in alfalfa. In contrast, immunity to *Phytophthora* is unknown (Marks and Mitchell, 1971b; Erwin, 1962) and resistance to the fungus has been increased over cycles of selection (Frosheiser and Barnes, 1973; Hine *et al.*, 1975).

b. *History* Goettel (1962) first discovered poor nodulation and poor growth of alfalfa in certain fields west of Edmonton, Alberta. Major fertilizer amendments did not substantially improve dry matter yield of alfalfa planted in sick soil over the alfalfa in unfertilized sick soil. There was a noticeable yield increase of alfalfa grown in fertilized, steam sterilized, sick soil compared to plants from a fertilized, nonsterilized, sick soil. In these greenhouse experiments, mention was made of the soil characteristics of the samples collected from alfalfa sick fields. However, as was with subsequent reports, no one characteristic of the soil could be identified as the casuative agent.

Webster *et al.* (1967) concluded that a biological toxic agent was responsible for the depressed growth of alfalfa plants in sick soil. Webster and his associates eliminated soil fertility, moisture deficiency, low pH or any combination of these factors as being the casual agent. In their study, plants from steam sterilized soil produced high dry matter yields. Certain alfalfa plants, which were grown in Leonard jars with aqueous soil extracts from various locations, showed a tolerance or resistance to the sick soil.

Goplen and Webster (1969) attempted to show that a genetic basis for selection of resistance to alfalfa sickness existed. However, the authors were unsuccessful, and part of this failure may have been a result of the selection criteria. The criteria relied on picking vigorous, healthy plants and adjacent sickly plants within an alfalfa-sick area and comparing their progenies. The selection procedure was at best superificial.

Webster and Dekock (1970) decided to examine field soil and plant samples for nutrient content. Nitrogen metabolism of alfalfa was affected by sick soil which had been previously cropped to alfalfa. An induced nitrogen deficiency and possibly an aluminum toxicity were suggested as two factors which affected the alfalfa in sick soil.

McElgunn and Heinrichs (1970) tried to assess fertilizer and root zone temperature effects in relation to alfalfa sick soil. Soil temperatures of 10°C, 15°C, and 20°C did not result in any significant differences for height and dry matter yield of alfalfa. Fertilizer interactions with alfalfa sick soil were not significant for any of the characters measured. Thus McElgunn and Heinrichs (1970) agreed with Webster et al. (1967) that nitrogen and phosphorous fertilizer did not alleviate alfalfa sickness. Root zone temperature provided no further clues as to the identification of this anomaly.

The Agriculture Canada (1972) report from the Swift Current Research Station concluded that since little progress was made in determining the cause of alfalfa sickness and year-to-year variation in severity of sick soil was great that continued study could not be justified.

Webster et al. (1972) initiated a survey of the nematode population of Paratylenchus projectus Jenkins since consistently high counts of this particular species had been observed in alfalfa sick soils. The highest counts of nematodes were recorded in dark gray luvisolic soils where alfalfa sickness was prevalent. It appeared the nematode was associated with sick soils. To substantiate this finding Webster and Hawn (1973) undertook a more extensive survey to determine the density and distribution of P. projectus, but were not able to establish a relationship between the nematode and alfalfa sickness. Certain soil parameters and cropping history were not correlated with the nematode counts.

When alfalfa was infected with P. projectus, no disease symptoms typical of alfalfa sickness could be induced (Damirgi et al., 1976). It was found dilutions of nonsterilized sick soil from 10^{-1} to 10^{-3} were required to incite alfalfa sickness symptoms. The dilution level was suggested to be correlated with infection and disease severity in the field. Thus Damirgi and colleagues were of the opinion that a microbiological agent was inciting alfalfa sickness.

Several points are evident. First, fertilizer treatments do not encourage alfalfa growth in sick soil. This means soil nutrients appear to be adequate and soil fertility is not the cause of alfalfa sickness. Second, soil moisture and soil pH have been eliminated as causative agents. Third, the cause of alfalfa sickness is biological. Sterilization treatments of sick soil, resulting in increased alfalfa yields, would tend to support the above statement. Finally, in relation to alfalfa sickness in the field, the symptoms appear on alfalfa sown in fields which had previously grown alfalfa.

c. Hosts *P. megasperma* was first discovered causing damage to alfalfa (*Medicago sativa*) by Erwin (1954b). The pathogen was found to be specific to alfalfa (*Medicago falcata* (diploid, tetraploid), *M. arborea*, *M. glutinosa*, *M. lupulina*) (Erwin, 1954a). Roots of cotton, sugar beet, aster, tomato, vetch, carrot, flax, sweetclover, ladino clover, red clover, bean, cowpea, and trefoil were not rotted by the fungus. Other species of *Phytophthora* were not pathogenic to alfalfa. Pratt and Mitchell (1975) reported oats, clover, corn, peas, and soybeans did not increase infective activity of P. megasperma . Johnson and Morgan (1965) when infecting garden pea, common vetch, and alfalfa (Medicago sativa cv. Buffalo and Delta) with P. cryptogea noted these plants to be susceptible. Frosheiser (1968) listed vetch, garden pea, sweetclover, cowpea, bean, and red clover as having been artificially infected. Numerous authors have also indicated that P. megasperma is highly virulent to alfalfa but did not test the infectivity on other plants. The range of hosts to which this pathogen can infect seems very narrow and specific to alfalfa.

Alfalfa sickness did not infect the roots of white clover, red clover, zigzag clover, alsike clover, sweetclover, birdsfoot trefoil, crown vetch, flax, or oats in naturally infected soil (Bolton, 1977). Roots of barley, milkvetch, sainfoin, and alfalfa were affected. However, Webster et al. (1967) found barley was not sensitive to the agent depressing growth of alfalfa in sick soils. Cormack (1940) found P. megasperma , isolated from sweetclover roots, was nonpathogenic to alfalfa. McIntosh (1966) isolated P. megasperma from irrigation sources in central British Columbia. However, the fungus was not pathogenic on alfalfa. Thus Phytophthora sp. have been isolated in western Canada although no infection on alfalfa has been induced.

d. Distribution Damirgi et al. (1976) stated alfalfa sickness was most prevalent on dark gray luvisolic soils in central Alberta. McKenzie and Davidson (1975) characterized several root and crown rots of alfalfa in the Peace River region of Alberta and British Columbia. No causal organisms were isolated and identified from the alfalfa root rots. However, the symptoms were described and attributed to several fungi one of which was P. megasperma .

In eastern Canada, Chi and Childers (1966) reported the association of several fungi with root rots of alfalfa in a disease survey over four years. P. megasperma was isolated from the cortex region of young alfalfa roots. Chi (1970) was able to isolate the fungus from alfalfa in 21 counties (76% of the fields sampled) in Ontario and Quebec.

In Washington, a condition known locally as alfalfa sickness has been described (Weber and Leggett, 1966). The cause was thought to be ineffective or insufficient Rhizobium meliloti for nodulation. Results from Elliott et al. (1968, unpublished) contradict Weber and Leggetts' findings by definitely indicating that insufficient or ineffective rhizobia are not the cause of alfalfa sickness.

Phytophthora root rot of alfalfa caused by P. megasperma has a wide geographic distribution. Areas reporting its presence include: California (Erwin, 1954b), Australia (Purss, 1959), Illinois (Bushong and Gerdemann, 1959), Ohio (Schmitthenner, 1964), Mississippii (Johnson and Morgan, 1965), Ontario (Chi, 1966), Minnesota (Frosheiser, 1967), Wisconsin (Marks and Mitchell, 1970), Arizona (Hine et al., 1972), Washington (Elgin et al., 1972), and North Carolina (Welty and Busbice, 1976). The common feature in these isolations has been the association of the pathogen with heavy soils, poorly drained or heavily irrigated, or with periods of above average rainfall.

e. Importance The pathological importance of the *Phytophthora* genus is far reaching. For example, this genus comprises nearly 70 reported species of plant pathogens which are nonobligate parasites of higher plants (Erwin *et al.*, 1963). Some of the diseases caused by *Phytophthora* sp. include: late blight of potato and tomato (*P. infestans*); red stele of strawberries (*P. fragariae*); wilt and root rot of avocado (*P. cinnamomi*); and root rot of soybean (*P. megasperma* var. *sojae*).

f. Symptoms Erwin (1954b) describes the symptoms of root rot of alfalfa caused by *P. cryptogea* as:

1) brown to red necrosis, irregular in shape, mostly occurring on the tap root but crown and lateral roots may be affected,
2) internally infected areas such as xylem may become water-soaked eventually becoming discolored and turning yellow, and
3) leaves lose their turgidity turning yellow and the roots become completely girdled.

Erwin (1954b) did not mention any effect of the disease on nodulation. Erwin (1965) later reclassified *P. cryptogea* to *P. megasperma* . Bushong and Gerdemann (1959) and Marks and Mitchell (1970) described the symptoms of Phytophthora root rot of alfalfa caused by *P. megasperma* . Marks and Mitchell (1970) felt this was the same pathogen that incited a cambial root rot of alfalfa reported by Jones (1943).

In Australia, Purss (1959) listed the symptoms of a root rot of alfalfa caused by P. cryptogea and P. parasitica . However, Irwin (1974) isolated a casual organism of Phytophthora root rot of alfalfa in Queensland, Australia which was P. megasperma var. sojae. This was in contrast to Purss's (1959) identification. A remarkable similarity exists among the symptoms described by Erwin (1954b), Purss (1959), Marks and Mitchell (1970), Bushong and Gerdemann (1959), and Damirgi et al. (1976).

g. Infection In all these accounts of P. megasperma and alfalfa sickness, the primary infection sites have been the lateral roots and eventually the tap roots. Scott (1965) differentiates lateral roots which form secondary thickenings from filamentous or transitory roots which may thicken slightly and have root hairs. Jones (1943) describes two types of roots in alfalfa based on the development of secondary growth. Permanent, cambial, or nontransitory roots form a cambium and phellogen whereas transient or noncambial roots develop very little, if any, phelleum and cambium. Thus lateral roots may be cambial or noncambial while the tap root is strictly cambial.

In the case of P. megasperma , Marks and Mitchell (1970) and Marks and Mitchell (1971b) suggested the lateral roots were most susceptible to infection. Zoospores (Marks and Mitchell, 1971a) of the fungus encysted on the root tips in the zone of cell division and cell elongation. The fine roots or noncambial roots were killed and lesions appeared on the tap root from which these fine roots emerged. Leakage of nutrients at this point was suggested to incise pentration and infection by the pathogen. This invasion and lesicn development on the roots led to reduced growth. As severity of infection increased chlorotic foliage, premature defoliation, and wilting occurred.

Irwin (1976), describing root infection of alfalfa by P. megasperma var. sojae, felt the junction of the lateral and tap root as well as the root tip were infection sites. The lateral root, in this case, would likely be transitory since the roots were examined 3 weeks after germination of seed. This suggested tap root lesions and loss of the noncambial roots under field conditions. A tactic response of zoospores was evident in the cultivars Lahontan, Hunter River, and Moapa. It appeared resistance to the disease in Lahontan occurred after host pentration by zoospore germ tubes.

In contrast, Gray and Hine (1976) thought early root infection in the field was associated with the nodules of alfalfa. From the nodule, P. megasperma could progress into the cortical root tissue and girdle the roct at the point of attachment of the nodule. But the roots from infected field plants were examined 4 months after seeding. This would seem somewhat late to justify that infection cf nodules, root tips, and nontransitory lateral roots had been observed.

The chemotaxis of zoospores to the host as suggested by

Marks and Mitchell (1971a) and Irwin (1976) is well documented in other Phytophthora caused diseases. Dukes and Apple (1961) observed a chemical attraction of zoospores of P. parasitica var. nicotianae to wounded tobacco roots. The substance causing chemotaxis was not specific to tobacco. Goode (1956) working with P. fragariae zoospores noted a tactic response of the zoospores to strawberry root tips. Zentmyer (1961) discussed chemotaxis of P. cinnamomi zoospores to avocado roots. Zoospores were attracted to the region of root tip elongation. A root exudate produced by the living avocado roots attracted the zoospores. Mehrotra (1970) using a fluorescent dye learned that zoospores of P. megasperma var. sojae accumulated on resistant and susceptible varieties of soybean seedlings. Thus, this phenomenon of chemical attraction of the pathogen to the host is supported by evidence within the Phytophthora genus.

The substance which attracts the zoospores of P. megasperma to alfalfa roots may be an amino acid such as thiamine. Data presented by Erwin and Katznelson (1961) tends to support the observation that thiamine may be important for survival of P. megasperma in the soil. Furthermore, microorganisms may be producing thiamine in the rhizosphere of the alfalfa roots.

h. Environment The single most important factor in the survival P. megasperma and its infection of alfalfa is moisture. Johnson and Morgan (1965) were able to isolate P. megasperma after heavy rains of 24.4 cm in April, 1964.

Frosheiser (1969) also isolated the pathogen during June, 1967 when 17 cm of rain was recorded with continous rain for 12 consecutive days in the midwest U.S.A.. Bushong and Gerdemann (1959) observed P. megasperma was favored by cool, rainy weather and standing water. The pathogen was frequently isolated in the spring and fall. Soil moisture levels below -40 bars reduced infective activity to nil (Pratt and Mitchell, 1975). At soil moisture levels near -15 bars, infective activity declined only slightly after 7 months. Also, irrigation increased disease incidence (Pulli and Tesar, 1975) of P. megasperma . Irwin (1974) mentioned the relation of rainfall and the presence of P. megasperma var. sojae in poorly drained soils. Lueschen et al. (1976) felt that 10 days or longer of saturated soil would cause rapid development of P. megasperma . Poor internal drainage of the topgraphy may also lead to increased inoculum levels of P. megasperma. However, when dry soil conditions result, detection of the pathogen may fail.

Temperature which favors disease development and expression was nonsignificant over a range of 17°C to 27°C (Erwin, 1966). This correlated well with the growth of P. megasperma in vitro. However, Pratt and Mitchell (1976) examining tap roots of resistant and susceptible selections found disease to be more severe at 20°C and 24°C compared to 16°C or 28°C. Gray and Hine (1976) indicated optimal soil temperature occurred at 23 cm below the soil surface June through August for high disease severity. Gray et al. (1973)

added that a temperature of 30-18°C or 24-13°C for light cycle (12 Hours) and dark cycle (12 hours), respectively, caused more disease than 35-24°C.

Inoculum levels of one-eighth and one-thirtysecond (v/v) gave the most severe disease to seedlings inoculated at 6 weeks of age (Pratt and Mitchell, 1976). These authors (Pratt and Mitchell, 1976) also stated dilution levels of 1:8 or 1:16 (original:diluent soil) allowed easy detection of _P. megasperma_ . Planting the infected soil to alfalfa selectively raised inoculum levels to make detection somewhat easier (Pratt and Mitchell, 1975). Nonhost crops did not raise the infective levels. Consequently, low inoculum levels in the soil may not be detected by current techniques. Damirgi _et al._ (1976) found alfalfa sickness more severe with 10^{-1}, 10^{-2}, and 10^{-3} soil dilutions compared to 10^{-4} and 10^{-5}.

Age at which alfalfa plants are attacked by the pathogen varies. Post-emergence damping-off was evident according to Johnson and Morgan (1965), Schmitthenner (1964), Bushong and Gerdemann (1959), Welty and Busbice (1976), and Gray _et al._ (1973) in greenhouse studies. Conflicting reports exist for pre-emergence damping-off. In support of pre-emergence damping-off, Welty and Busbice (1976), Gray _et al._ (1973), and Bushong and Gerdemann (1959) reported it but Schmitthenner (1964) and Johnson and Morgan (1965) found no evidence in the greenhouse to support this view.

Frosheiser (1967) found disease severity of *P. megasperma* did not differ on alfalfa from 1 to 6 weeks of age. At 12 weeks, the disease severity (5.9) was less than 1 to 6 weeks (7.5-8.1). Welty and Busbice (1976), testing the pathogenicity of *P. megasperma* over time, discovered alfalfa inoculated at 21 days of age had the root completely rotted. Plants inoculated at 28, 35, and 50 days had necrotic root lesions, stunting, and yellowing. When inoculated at 71 days of age, only yellow lesions developed on the foliage of alfalfa. Gray and Hine (1976) indicated initial field infection of alfalfa roots may occur 4 to 8 weeks after seeding. This agrees with Lueschens' *et al.* (1976) results in which yield reductions due to *P. megasperma* damage were suspected to occur during the seeding year. Purss (1959) stated first year stands of alfalfa are more susceptible to attack than established stands.

The survival of *P. megasperma* may be in the form of oospores (Frosheiser, 1968) or chlamydospores (Schmitthenner, 1970) since other structures would be killed by environmental stress, most notably drought. This is supported by Tsao's (1969) work on *P. parasitica*. The primary infective propagule of *P. megasperma* is the zoospore and suspensions of it have been used for inoculation studies in the greenhouse (Marks and Mitchell, 1975). But most investigators have utilized mycelium both in greenhouse (Gray *et al.*, 1973) and field experiments (Frosheiser and Barnes, 1973; Lu *et al.*, 1973).

The fungus is capable of surviving in the soil at various soil depths. Frosheiser (1969) observed tap root rotting 50 cm below the soil surface. Gray and Hine (1976) detected root lesions from 4 to 40 cm below the soil surface but noted some as deep as 80 cm. Soils prone to root rot of alfalfa caused by _P. megasperma_ are silty loams and podzolized (Marks and Mitchell, 1970). Purss (1959) suggested heavy soils with an impervious layer were conducive to spread of _P. parasitica_ and _P. cryptogea_.

2. Plant

Foliage diseases of forages caused an estimated annual loss of 5.9% or 6.2 million dollars from the period 1970-1973 in central and northern Alberta (Berkenkamp, 1974). Alfalfa disease losses in the U.S. have been estimated at 24% for forage and 9% for seed (Graham _et al._, 1972). In the U.S., root and crown rot fungi cause damage to at least 5% of the alfalfa. The only practical means to overcome these losses are through control by the use of crop rotation, chemicals, or resistant cultivars.

a. _Management_ Crop rotation was not effective in reducing the level of infective activity of _P. megasperma_ (Pratt and Mitchell, 1975). The crops included in the rotation were corn, oats, clover, soybeans, and peas grown for 3 to 6 months. In this greenhouse study, infective activity in soil planted with the above crops was compared to infective activity in fallow.

Crop sanitation has been practiced to rid an area of the source of infection. Bonde (1943) documented burning potato cull piles to eliminate the infection source of late blight of potato caused by *P. infestans*. Turner (1965) suggested removing and disposing of cacao pod material in the field. This acts as an infection source for *P. palmivora* . Crop removal by burning has not been reported for controlling the dispersal of *P. megasperma* .

Frequent irrigations will increase disease severity of *P. cinnamomi* (Zentmyer and Richards, 1952). These weekly waterings of avocado trees meant the disease appeared sooner and caused greater damage. Pulli and Tesar (1975) stated that when 10 cm of water was added in addition to the normal rainfall, the disease incidence and severity of *P. megasperma* on alfalfa increased. Other management factors which stressed the alfalfa plants making them susceptible to attack by *P. megasperma* were: increasing cutting frequency from 2 to 3 times per year, seeding at higher rates (9 to 36 kg/ha) and late seeding (April 27 compared to May 19). Thus, mismanagement will increase the likelihood of infection of alfalfa by *P. megasperma* and result in lower yields.

b. *Chemicals* The application of fungicides to control root diseases of alfalfa has been limited due to the cost of chemicals and their lack of effectiveness over a number of years. For example, the only disease in which a fungicide was cited for disease control in alfalfa by Graham *et al.* (1972) was spring black stem and leafspot caused by *Phoma* sp. Even at that, the chemical was uneconomical and the residual effect was unknown. Fungicides such as Dexon (sodium 4-dimethylaminophenyl diazo sulphonate) have been used to control fungi in the Phycomycete class (Kreutzer, 1963).

The chemical, Dowco 269 (2-chloro-6-methoxy-4(trichloromethyl)pyridine) or pyroxchlor, was inhibitory to *P. megasperma* , *P. cryptogea*, *P. cinnamomi*, and *P. megasperma* var. *sojae* according to Hoitink and Schmitthenner (1975). Dexon was not effective in controlling rhododendron root rot caused by *P. cinnamomi* or root rot of soybean caused by *P. megasperma* var. *sojae*. But Dowco 269 applied as a drench before disease inoculation controlled *P. cinnamomi* in the greenhouse. When applied as a fungus treatment, the chemical restricted soybean root rot but some stunting of the plants occurred. Dowco 269 did not kill *Phytophthora* sp. in vitro or eradicate *Phytophthora* from diseased plants.

Stuteville (1976) applied Dowco 269 to alfalfa seeds and evaluated seedling reaction to *P. megasperma* . The fungicide was phytotoxic at the 2% level (based on weight/weight basis and expressed as a percentage of the seed weight). Dowco 269 increased the percentage of living plants of the Phytophthora-resistant cultivar Agate. No deleterious effects were observed on nodules of alfalfa. This appears to be a promising fungicide for control of *P. megasperma* . But Dowco 269 is only preventative in its action against the pathogen.

c. Plant Breeding The primary consequence of selection is to change the frequencies of genes affecting the trait upon which selection acts. In this regard, the genetic properties of a population can be altered by the choice of individuals to be used as parents and this constitutes selection. However, the initial gene frequency, rate of change in gene frequency, and mode of gene action will determine the magnitude and degree of selection response. Consequently, Allard (1960) has characterized five selection response patterns.

Robertson and Reeve (1952) studied the inheritance of wing and thorax length in *Drosophila melanogaster* over 50 generations. The phenotypic variance of the characters under selection increased as an indication of response to selection. Sheldon (1963), in discussing selection response for body weight in *Drosophila melanogaster* over 39 generations, noted response to selection continued, not reaching a plateau, in the low lines. In the high lines, the response was absent over the selection period since culture changes and genotype by environment interactions occurred. Falconer (1953) described the response to selection as proceeding regularly, showing no sign of plateauing in a selection experiment for mouse size over 11 generations. Woodworth *et al.* (1952) presented data on fifty generations of selection for protein and oil in corn. The response was slow and steady with a gradual shifting of the population means in both the high and low protein and oil. For each of these studies, except Falconer (1953), the major divergence between high and low lines for the characters selected generally began at generation five. In all the studies, genetic variability did not decrease and the slow, steady response to selection over a number of generations is the first such selection pattern (Allard, 1960).

The lack of response to selection in corn for lower oil content after 25 generations (Woodworth *et al.*, 1952) was thought to be due to a physiological threshold. This did not imply fixation of the genes for low oil content. There also may be a complete lack of response to selection as was suggested by Allard (1960). The example mentioned by Allard (1960) was selection for yield in corn and the failure of selection was attributed to the low heritability of this character.

Mather and Harrison (1949) illustrated another type of selection response in *Drosophila melanogaster* for abdmonial chaetae over at least 100 generations. This type of response was in the following form; gain from selection followed by a plateau in which there was no response to selection, then a gain in selection followed by a plateau. The authors concluded there was a large amount of hidden genetic variability which provided the organism with flexibility or potential variability to survive natural selection of overall fitness. This flexibility was counterbalanced by free genetic variability which was released by segregation as a result of directional or artificial selection.

The last type of response to selection is one which is most likely to occur in breeding resistance to diseases. The response is initially rapid followed by a period of slow progress (Allard, 1960). The reasoning thought to explain this response is fixation of a small number of genes which have been referred to as major genes. The slow progress results when the major genes have been selected to a point of fixation and only changes in the frequency of minor genes occurs. This pattern of selection response is common in inheritance of resistance and susceptibility to most fungi diseases of wheat, barley, corn, and flax.

As a result of the control exerted by major genes for disease resistance, Flor (1959) proposed a gene-for-gene hypothesis which assumes host and pathogen have evolved together. In relation to diseases cf alfalfa, this hypothesis has not been applied.

Phenotypic recurrent selection as a method of selection has been widely used to breed resistance to disease in alfalfa (Twamley, 1974). The method consists of selecting desirable clones, intercrossing the selections, and using the seed for another cycle. The term phenotypic relates to resistance being controlled by few genes which are highly heritable. Hanson _et al._ (1972) summarized the results of a program using recurrent phenotypic selection for conserving germplasm and developing disease resistance. This selection method proved successful for Hanson and his associates.

Two programs were initiated, one at St. Paul, Minnesota by Frosheiser and Barnes (1973) and the other at Tucson, Arizona by Gray _et al._ (1973) to develop resistance to _P. megasperma_ . A greenhouse seedling selection technique involving phenotypic recurrent selection and polycross progeny evalution was utilized by both groups. Environmental effects were small, heritability high, and gene recombination favorable in both selection procedures. This was indicated by the response to selection. Frosheiser and Barnes (1973) stated the level of resistance went from 10% initially to 50% after 2 cycles of selection to 63% after 3 cycles. Hine _et al._ (1975) also increased the percentage of resistant plants from 15% initially to 60% in 1 cycle to 83% after 2 cycles of selection. Thus, phenotypic recurrent selection in the greenhouse effectively increased resistance to _P. megasperma_ .

Alfalfa disease resistance in most of the studies discussed by Kehr _et al._ (1972) has been conditioned by a simply inherited tetrasomic gene. Resistance to _P. megasperma_ in alfalfa was also found to be governed by a tetrasomically inherited gene with incomplete dominance (Lu _et al._, 1973). Accumulative gene action determined the genotypes as follows: highly resistant=nulliplex genotype; moderately resistant=simplex genotype; and increasing susceptibility=duplex, triplex, and quadruplex. The same gene which controlled resistance in alfalfa of Turkestan origin controlled resistance in winter-dormant cultivars such as Vernal. This simply inherited trait was eludicated using a diallel, selfing, and testcrossing so that 3 generations could be observed simultaneously. Heterozygous parents and a small family size of 30 allowed the scheme to be quickly completed.

The mode of resistance to P. megasperma was examined by Marks and Mitchell (1971b). Two factors appeared to be associated with the resistant reaction which were; first, the structure of the central stele and second, a hypersensitive reaction observed in cortical cells of growing root tips. Increased lateral root numbers with larger steles and clearly defined endodermis contributed significantly to resistance. Secondly, a granular phenolic compound was observed in young, living cortical cells of tolerant cultivars. This material might possibly be a phytcalexin as has been reported in soybean (Keen, 1971) in the resistant reaction to P. megasperma var. sojae. This hypersensitive reaction was confirmed by Pratt and Mitchell (1975) inoculating resistant and susceptible alfalfa plants with zoospore suspensions of P. megasperma . Small, local, necrotic lesions developed in resistant plants suggesting hypersensitivity. In susceptible plants, tissue collapse and death were observed in roots, cotyledons and leaflets.

Cultivars resistant to P. megasperma have been developed and one such cultivar is Agate (Medicago sativa) developed by Frosheiser and Barnes (1973). The average disease severity index of Agate (tested as MnP-A2(Syn. 1)) was 2.40 with 63.5% resistant plants. Compare with this, Vernal at 3.87 and 6.2%, Saranac at 3.95 and 5.5%, and Lahontan at 2.92 and 36.3% for disease severity and per cent resistant plants, respectively. The disease severity was a scale of 1 to 5 with 1=no symptoms and 5=plant dead. The per cent resistance was based on plants scoring a 1 and 2 disease rating.

Lehman *et al.* (1969) has licensed 2 germplasm sources UC38 and UC47 with tolerance to *P. megasperma* . The degree of tolerance, based on a disease rating (0=no disease and 5=completely rotted root) and the percentage tolerant plants with a disease rating cf 2 or less, were 3.4 and 23.1% for UC37 and 2.9 and 38.3% for the check Lahontan, respectively. In another test, the germplasm UC47 had a disease rating of 4.4 and 14.3% tolerant plants compared to Lahontan with 2.9 and 32.1% tolerant plants.

A germplasm release from the Pullman Washington Agricultural Station by Elgin (1977) documents the percentage resistant plants of WDS3P1 to Phytophthora root rot. There were 64% resistant plants in WDS3P1 based on one cycle of greenhouse selection; 6% in Vernal; 47% in Agate; and 2% in Saranac. Per cent resistance was determined by the number of plants scoring a 1 and 2 disease rating.

Comparison of various cultivars at the St. Paul, Minnesota nursery (Frosheiser, 1977) for *P. megasperma* resistance are summarized as follows; Agate-2.70, Grimm-4.86, Ladak-4.26, Lahonton-3.00, Rambler-4.18, and Vernal-4.56. The average disease severity index, following each

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cultivar, was based on a scale of 1 to 6 with 1 being no symptoms and 6 being a dead plant.

Various characters are measured and methods used in testing plant material for resistance to disease. For example, Graham _et al._ (1965) lists the advantages of making selections in the growth chamber compared to field selection. Caution must be exercised that any characters measured do not change when the plants are transplanted to the field. If the populations in which selection has been practiced are broad-based, no appreciable shift in other traits should occur. Hill _et al._ (1969) confirmed this observation and stated 75 or more plants per cycle of selection should be selected to prevent inbreeding depression.

In looking at the characters to study, Carnahan (1963) evaluated reciprocal differences in alfalfa for maternal effects in seedling height and yield. They appeared to be controlled by the nucleus rather than the cytoplasm.

Devine and McMurtrey (1975) correlated disease rating (1=low frequency, 9=high frequency of diseased plants) with stand count and yield in anthracnose resistant alfalfa lines. Disease rating was correlated from 0.06 to 0.65 with stand count over 2 years in the field. For yield, the correlation ranged from 0.01 to 0.56 in the field data. Theurer and Elling (1963) used a disease rating for bacterial wilt of alfalfa caused by _Corynebacterium insidiosum_. The rating was based on scale of 0 to 5 with each class representing varying degrees of disease symptoms. Class 0 was no evidence of wilt while 5 was a very severe infection. The phenotypic correlation (r=0.61) between each of the synthetics and their single crosses was highly significant.

Frosheiser and Barnes (1973) found significant correlations of r=-0.979 and r=-0.865 for forage yield and average disease severity index. The disease severity index was determined by an individual score for each plant (1=no symptoms, 5 and 6=dead plants) in the field. Lueschen _et al._ (1976) related forage yield to infection and injury caused by _P. megasperma_ as did Frosheiser and Barnes (1973). Lueschen and associates assumed in noninoculated field plots with natural rainfall that _P. megasperma_ was causing the damage. This damage was reflected in the lower yields of susceptible cultivars compared to the resistant lines.

Berkenkamp and Baenziger (1969) found percent survival, which was estimated visually, was correlated with lesioning of the roots (r=0.73) in sweetclover. The lesions were caused by _Plenodomus meliloti_ and the correlation was a indication of resistance to the pathogen.

Combining ability has been used as a method to evaluate alfalfa clones for their behavior in combination with other clones (Bolton, 1948). Sprague and Tatum (1942) stated general combining ability (GCA) represented additive genetic variation while specific combining ability (SCA) referred to dominance effects, epistatic effects, and genotype by environment interactions (Rojas and Sprague, 1952). These authors (Sprague and Tatum, 1942) defined GCA as the average performance of a line in hybrid combination and SCA as performance of certain crosses which do better or worse than the average performance of the lines. In unselected material, the GCA variance or additive effects were greater than the SCA variance or nonadditive effects. If lines are selected for high GCA, then the component of variance for SCA may exceed that for GCA. This is due to the greater degree of resemblance between selected lines compared to the unselected material.

The importance of GCA and SCA for diallel crosses of alfalfa depends upon the choice of parental clones. For unselected material, the GCA variance has been found to be more important than the variance for SCA (Pearson and Elling, 1960; Carnahan et al., 1962). In contrast, Singh and Lesins found the significant SCA variance was greater than the GCA variance and genotypic variance was largely nonadditive. But the clones evaluated in Singh and Lesins' (1971) study had been previously selected. The merits of both types of results are useful from the standpoint that clones with high GCA effects can be used to form a synthetic cultivar of a number of clones. Or, any 2 parents with consistently high SCA effects can be used to form a 2-clone hybrid.

Heritability estimates have been used to determine the relationship between the phenotype and genotype. These estimates are relative only to the populations and environmental conditions in which they were derived. Various procedures have been utilized such as analysis of variance and regression to estimate the amount of genetic variation. In the analysis of variance (Falconer, 1960), the genotypic variance which includes additive, dominant, and epistatic gentic variance is expressed as a ratio of the environmental or phenotypic variance. Preceding further, as in the diallel (Gardner, 1963), the analysis of variance provides estimates of heritability in the broad sense which includes additive, dominance and epistatic genetic variances and the narrow sense which is additive genetic variance.

. The second technique relies on regressing the offspring on the parent as proposed by Lush (1940). This method includes additive, digenic, and heritable portions of epistatic variance with appropriate coefficients and is a superior method for predicting gain from selection (Swanson et al., 1974). Swanson and colleagues considered parent-offspring regression as the best estimate of narrow sense heritability in an autotetraploid such as alfalfa.

Broad sense heritability estimates are usually high in alfalfa as Dudley et al. (1963) reported them to be 59% to 80% for resistance to rust in alfalfa. Carnahan et al. (1962), in a study of common leafspot resistance in alfalfa, found the broad sense heritability to be 64%. Devine et al. (1971) stated resistance to anthracnose in alfalfa was highly heritable. These estimates of broad sense heritability imply genotypic variability exists and progress in selection can be accomplished.

MATERIALS AND METHODS

1. Materials

The materials used as sources of resistant and susceptible genotypes were cultivars of *M. media* -Beaver (B), Grimm (G), Roamer (R), and Vernal (V). Susceptible genotypes were designated by 'P'. Cycle 0 refers to the unselected plants while cycle 1, 2, and 3 refer to three cycles of selection for resistance or susceptibility to alfalfa sickness. For example, 2VP16A would designate a Vernal plant or progeny in the second cycle of selection that is susceptible. The 16A is an arbitary number given the original genotypes from cycle 0. In the growth room tests, Beaver was the check cultivar used. The cultivars which acted as checks in all the field tests were Beaver, Grimm, Roamer, and Vernal. The check cultivar Agate (Barnes *et al.*, 1973) was included in strain test 1 and the observational strain test. The cultivars in the field were replicated eight times in lattice strain test 1 and 2 while in strain test 1, the cultivars were replicated four times. This increased replication of the cultivars provided a more accurate comparison with the genotypes.

2. Methods

To achieve our objectives, eight growth room tests were undertaken to study the control of the alfalfa sickness pathogen by chemical and physical treatments. Three growth room tests and five field tests were directed at examining the control of the alfalfa sickness pathogen by plant breeding. The tests, their location, replication, and experimental design are listed in Table 2.

a. Growth Room The growth room in which the tests were undertaken was maintained at approximately 18°C. Lighting for 20 hours per day was provided by florescent bulbs which supplied about 21,500 lux at approximately 45 cm above the pots. Plastic pots were closed at the bottom to prevent the loss of any possible toxic substance which might be associated with the disease. The pots were 17 cm in diameter and contained about 1,800 g of soil when filled to about 2 cm below the rim.

The bioassays utilized in the growth room tests were sick soil and isolates of organisms from sick soil. Sick soil was obtained from areas known to induce alfalfa sickness; namely, an area near Breton, Alberta, and later, a site at Spruce Grove, Alberta selected for the field tests. Uninfected soil was collected from the Edmonton Research Station (Parkland Farm); and a virgin soil from Breton, Alberta. Individual soil samples were taken to a depth of 15 cm below the surface from several locations within a sixteenth of a ha, and were combined. This composite sample was passed through a 0.6 cm wide mesh screen, and then was thoroughly mixed and stored in plastic bags until needed. A portion of the composite sample which was at field moisture capacity was steam pasteurized at 1.2 kg per cm² for 30 minutes at 120°C, and stored in plastic bags. Fertilizer was added before potting by mixing 1.5 g of 10-30-10 with the soil in a plastic bag. Commercial inoculum of *Rhizobium meliloti* was spread on the soil surface of the pot immediately after planting. The field moisture capacities were determined and these values were used to decide the amount of water to add to each pot before planting. After planting, water was applied every second day to bring the pots back to field capacity. Sick soil used in the time of infection test, fungicide test 1, and second cycle strain test was collected from the field in November, 1975. Sick soil for the other growth room tests was obtained in July of 1976 except for the diallel strain test which was collected in October, 1976.

The second type of bioassay, used only in the inoculation test, was isolates, labelled F6, F9, and F16, of pathogens responsible for alfalfa sickness according to Damirgi *et al.*, 1976. Stock cultures of *Phytophthora megasperma*, numbers 446, 844, and 892 were obtained from Erwin (1976). Mats of the isolates and stock culture were grown on potato dextrose agar medium for 8 to 10 days and then removed, pooled, and water added. The mixture was ground in a blender to form a paste of which forty mls of this paste inoculum was poured on the moist surface of pasteurized soil in each pot.

Lime, in the form of finely ground calcium carbonate, was added to those soil samples that showed an acid reaction. Such treatment was given to soils from soil tests

1 and 2, fungicide tests 1 and 2, and the second cycle strain test. The application rate was determined by weighing 50 g of air-dry soil into a waxed paper cup, adding lime and mixing well, and bringing to a moist capacity. The soil was left for a week before the pH was determined. Treatments for each soil in the growth room tests listed above were no lime (control); 720 kg per ha; 1440 kg per ha; 2160 kg per ha; and 2880 kg per ha. These liming rates were intended to correct the acidity by raising the pH to 7.0. However the high rate of lime (2880 kg per ha) was not always sufficient to increase pH to neutrality. To overcome this problem a graph of the lime rates and pH values was plotted so that the lime rate could be extrapolated to pH 7.0. Thus, soil moisture, soil fertility, and pH were corrected to optimum levels so as not to confound response to these factors with response to alfalfa sickness.

The growth room tests involved germinating scarified alfalfa seeds on moist filter paper in petri dishes. Fifteen to seventeen seedlings per pot were planted when the radicle was half a centimeter in length. The tests were terminated at 35 to 40 days when the alfalfa seedlings were 15 to 40 cm in height.

b. Field The area chosen for field tests was selected on the basis of data from soil test 1 indicating alfalfa sick soil. The field site, 774 meters above sea level, is approximately 16 km west of Edmonton, Alberta on the south east quarter of 16-53-27 W4. The soil type is a chernozem which is within the black great group (Bowser et al., 1962). It is a sandy loam composed of 75% Peace Hills fine sandy loam and 25% Ponoka loam. The topography of the land is undulating and depressional with a slight knoll sloping to the north. Previous cropping history of this area was oats threshed for grain for the past 3 years. The preceding crop was alfalfa which was cut for hay. The alfalfa stand was 5 years old when it was decided due to a declining, unthrifty stand that it should be plowed under in favor of a more profitable crop.

Physical characteristics of the soil selected for the field site are tabulated in Appendix 1. The field area was sampled on August 18, 1976 and the soil samples marked 'J' were collected from an area immediately adjacent to the field site. This area adjoining the field site had been fertilized with 67 kg per ha of 11-55-0 before alfalfa was undersown with a crop of barley. The nitrogen and phosphorus levels were adequate but potassium was low for sample numbers 1, 11, and 12. Sulphur was moderately low for most of the soil samples. No free lime or sulphates were detected and only a trace of sodium was found in all samples. The pH of the soil samples was slightly acidic.

Weather data were analyzed for the past 10 years for the field study site at Spruce Grove, Alberta (Appendix 2). The average rainfall was 30.5 cm for May through August while the mean temperature was 14.8°C for the same period. Simple correlation coefficients between rainfall and temperature were low and not significant (r=-0.21 to 0.06). Correlations between temperature and time (r=-0.44 to 0.09) were also nonsignificant but the correlation between rainfall and time (r=0.77) was highly significant.

The field site was double disced, harrowed, rotovated, and packed prior to seeding. To eliminate fertility effects, 224 kg of 11-55-0 per ha and 224 kg per ha of 0-45-0 were broadcast with a cyclone spreader on May 13, 1976.

The alfalfa seed was scarified and commercial inoculum of _Rhizobium meliloti_ was added to the seed before planting. Hand pushed V-belt seeders were used to sow the seed to a depth of one and half cm at 25 seeds per 30 cm on May 14, 1976. Water was applied by sprinkler irrigation May 28, 1976 while hand weeding of the plot was undertaken on June 14, 1976 and July 19, 1976. The plot was sprayed June 25, 1976 with MCPA amine 80 ((4-chloro-o-tolyl) oxyacetic acid) at 210 g active ingredient per ha.

The agronomic characters of alfalfa chosen for evaluation of the effect of the disease pathogen on the plant were the following:

1) plant height,
2) plant stand,
3) stand survival,
4) disease rating,
5) performance rating, and
6) yield.

Plant height in the growth room was measured in cm from the cotyledonary node to the tip of the plant's leaves for each plant. Height, in cm, was measured from the soil surface to the leaf tips in each row on September 9, 1976 and June 17, 1977 for all field tests. The plant height assessed in the field September 9, 1976 in strain test 1 was the mean of 2 measurements per single row.

The plant stand was a rating on all field tests August 3, 1976 using a ruler to determine the number of gaps 10 cm or larger per row. A score of 1 to 9 was used with 1 meaning no gaps or a complete stand and 9 indicating 9 or more gaps per row. This rating was used to adjust yield data taken from the field tests in 1976 and 1977. Stand survival in the growth room was a count of the number of plants per pot one week after planting for soil test 1.

The disease rating was a visual examination of the roots of each alfalfa plant under a stereo-microscope. The plants with roots intact were soaked about 15 minutes in a water bath, then the soil was washed off the roots with running water. The roots were rated on a scale of 1 to 5 with 1 being clean, healthy roots and 5 being a dead plant. The ratings 2, 3, and 4 were intermediate with 2 being roots with slight browning and lesions; 3, roots exhibiting brownish, well-defined lesions; and 4, browned, girdled roots with severe lesioning. This scheme of rating symptoms was based on other alfalfa disease studies (Frosheiser and Barnes, 1973; Gray _et al._ 1973). Rating classes 1 and 2 would indicate resistance while classes 3, 4, and 5 would be susceptible to alfalfa sickness. The disease rating was measured on all plants in each growth room test except soil test 1. For strain test 2, 10 randomly selected plants in each row were uprooted to a depth of 20 to 30 cm and rated in the field. The disease rating for strain test 2 was carried out on August 5, 1976.

The disease rating used in scoring root reaction to alfalfa sickness in cycles 0 and 1 in the growth room was different. No class 5 rating existed however the procedure followed was identical to the method of rating described above.

The performance rating for strain test 1 was a measure of the vigor, productivity, and general appearance of the stand in a row. The field rating was based on a scale of 1 to 5 with 1 being an unproductive, unthrifty stand and class 5 being a complete stand with vigorous, healthy growth. Ratings 2, 3, and 4 were intermediate classes of productivity and thriftness of the stand. An average of three individual, independent assessments of the stand in each row was taken on August 19, 1976.

Yield for growth room tests was measured on a grams per pot basis. The material was air-dried in paper bags for 48 hours at 48°C. For soil test 1 and the second cycle selection test, the material above the cotyledonary node was dried and weighed while in fungicide test 1 and the time of infection test the roots of the plants were used for yield. In the remaining growth room tests, the whole plant (top+root) was weighed for yield.

Strain test 1 was harvested for yield October 25, 1976 using a sickle mower while all the field tests were cut with a Mott mower in 1977. In 1976, the cut material from an entire row was placed in a cotton sack, air dried for 48 hours at 48°C, and weighed to the nearest gram. Due to the limited drying facilities and substantial green yield, a different procedure was followed when the field tests were cut on June 20, 1977. A 300 gm subsample of the weed-free green yield from each row was taken and dried for 48 hours at 48°C. The dry weight of the subsample was used to calculate the grams of dry matter per row. No winterkill was evident after the 1976-77 winter and establishment of the field site was successful.

c. Pathogen Soil test 1 was concerned with identifying an area affected with alfalfa sickness for a field site and studying physical control of the pathogen by heat and pH. The test was a split plot with the soil samples treated as main plots and the control treatments as subplots. The control treatments included pasteurization, pasteurized-limed, nonpasteurized, and nonpasteurized-limed.

The second soil test, soil test 2, was an assessment of the soil from the proposed field site for the presence of alfalfa sickness. The effect of pasteurization was also examined as a means of pathogen control. Five locations were sampled within the field site.

The U.S. soil test was an observation test in which soils from different geographic areas purported to be infected with P. megasperma were imported and evaluated at the Edmonton Research Station (Parkland Farm). A comparison of the symptoms exhibited by Beaver alfalfa infected with P. megasperma and alfalfa sickness was the object of this particular test. There were limited quantities of soil available to pot, making a replicated trial impossible. The soils from P. megasperma infected areas were: Ottawa, Ontario, Canada[1] ;East Lasing, Michigan, U.S.A.[2] ;St. Paul, Minnesota, U.S.A.[3] ;Davis, California, U.S.A.[4] .

To determine the initiation and progress of alfalfa sickness with a view for knowing when control would be appropriate, diseased roots were examined weekly over a seven week period. This was the purpose of the time of infection test. Regression techniques were used to predict the relationship between time and height, and time and disease rating. These functional relationships provided information about the effect of the disease on alfalfa over time.

The second bioassay method which consisted of isolates of the alfalfa sickness organism and P. megasperma was utilized in the inoculation test. This test was undertaken with the idea that a technique could be developed for evaluating alfalfa genotypes grown in inoculated soil in the growth room. No infection of alfalfa by the alfalfa sickness organism in potted soils had been investigated before this present test. Furthermore, a comparison of the response of resistant and susceptible genotypes in pasteurized sick soil was the third aim of this inoculation test. Two resistant (1V12, 2V96), and two susceptible (1GP130, 1RP188) genotypes plus Beaver were planted in the pasteurized inoculated and pasteurized noninoculated sick soil.

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[1] Chi, C.C. 1976. Research Branch, Ottawa Research Station, Ottawa, Ontario, Canada.
[2] Tesar, M.B. 1976. Department of Crop and Soil Science, Michigan State Univ., East Lasing, Michigan, U.S.A..
[3] Frosheiser, F.I. 1976. Department of Plant Pathology, Univ. of Minnesota, St. Paul, Minnesota, U.S.A..
[4] Stanford, E.H. 1976. Department of Agronomy and Range Science, Univ. of California, Davis, California, U.S.A..

The effects of chemical control of alfalfa sickness were investigated in fungicide test 1 and 2, and the strain + fungicide test. The fungicides included Benlate (benomyl), Dexon (fenaminosulf), Metazoloxon (drazoloxon), and Dowco 269 (nurelle). The chemicals were applied as drenches to the soil surface, mixed in with the soil, or both. Dowco 269 ((2-chloro-6-methoxy 4-(trichloromethyl)pyridine)) was also applied as a foliar spray.

Fungicide test 1 was a preliminary test of Dexon and Benlate applied as drenches. For fungicide test 2, Dexon, and Metazoloxon were applied to the sick soil by (1) mixing in with the sick soil, and (2) mixing in with the sick soil plus drenching; and for Dowco 269, it was applied to sick soil by (1) mixing in with the sick soil, (2) spraying on the foliage, and (3) spraying on the foliage plus drenching. This was to determine if the fungicides were effective against alfalfa sickness and if one application method was more satisfactory than another in controlling the pathogen.

Finally, the strain + fungicide test was a combination of 3 treatments applied to the cultivar Beaver, a resistant genotype (1V12), and a susceptible genotype (1GP130). The treatments were: (1) Dowco 269 applied as a drench to the soil surface, (2) a pasteurized soil check, (3) a sick soil check. As well as the chemical control of the pathogen, the genetics of the plant may be manipulated to produce a suitable combination of genes for inherited resistance to alfalfa sickness. Thus, control of alfalfa sickness by a combination of chemicals and heritable resistance may provide more protection against alfalfa sickness than either chemicals or plant breeding alone. The second intent of the strain + fungicide test was to compare resistant plant and susceptible plant responses under conditions which should and should not elicit the response. These conditions were sick soil and pasteurized sick soil, respectively.

The fungicide application rates and methods utilized in the above tests are described as follows. Benlate ((methyl-1(butylcarbamoyl)-2-benzimidazolecarbamate)), as a 50% wettable powder, was applied at the commercial recommendation of 10 ounces per 1000 ft^2. Dexon (sodium 4-dimethylaminophenyl diazo sulphonate), 70% wettable powder, was put on at the recommended rate of 10 ounces per 1000 ft^2 in fungicide test 1. In fungicide test 2, the rate was 10 g active ingredient (a.i.) per 6 litres of soil for mixing or

2 g a.i. per litre of water for drenching. The Metazoloxon, technical product, ((4-(3-chlorophenylhydrazono)-3-methyl-5-isoxazolone)) was applied at only recommended dosages due to the availability of limited quantities of the product. These rates were 0.18 g a.i. per litre of soil as a mix and 0.25 g a.i. per litre of water as a soil drench. Dowco 269 (12% a.i. emulsifiable concentrate) was put on the soil at 0.2 g a.i. per litre of water as a drench or foliar spray and 1 g a.i. per 6 litres of soil as a mix. The recommended application rates were determined according to Evans (1976).

The fungicide drench involved mixing the chemical with 500 mls of water, then pouring 100 mls of the solution on the soil surface of each pot. This was applied weekly (4 applications) except for Metazoloxon and Dowco 269 which were put on bimonthly (2 applications). Dexon and Benlate were drenched on the soil surface one week after planting whereas Metazoloxon and Dowco 269 were first applied 2 weeks after planting.

To prepare the fungicide mix, the soil and chemical were placed in a plastic bag an hour prior to planting. After thorough shaking the mixture was weighed back into each pot.

Dowco 269 was applied as a foliar spray by mixing the fungicide with 500 mls of water. One hundred mls was sprayed on the alfalfa foliage of each pot. The spray was applied twice starting 2 weeks after planting. No steps were taken to prevent drainage of the Dowco 269 from the foliage into the soil.

As an added note, Dexon and Benlate are registered products in Canada. Dexon has known activity against pythiaceous fungi whereas Benlate has no known activity against these fungi. Metazoloxon and Dowco 269 are experimental chemicals in Canada however Dowco 269 has subsequently been withdrawn as an experimental chemical (Evans, 1976).

d. Plant The criteria for selection in cycles 0, 1, 2, and 3 were height and disease rating. The reason for the choice of these two traits was due to observations (Bolton, 1977) that taller plants occurred among alfalfa sick plants and upon examination in the laboratory, these taller plants had less diseased roots than the plants which appeared sick. The seedlings in each cycle of selection were evaluated at 35 to 40 days of age and eight populations in total for cycle 1 were established to represent resistant and susceptible groupings of the 4 cultivars originally used in cycle 0. For cycles 2 and 3, cultivars were disregarded to give only 2 populations, one resistant and the other susceptible. Genotypes from the populations were maintained in the growth room to the flowering stage, and polycrossed within each population by transferring pollen on a toothpick from plant to plant. At maturity the pods were harvested, threshed, and stored. The parent plants from the selection program were transplanted into a reserve field nursery at the Edmonton Research Station (Parkland Farm).

The second cycle strain test was used to evaluate genotypes of cycle 2 and to initiate a third cycle for further testing. This test included 59 resistant genotypes, 23 susceptible genotypes, and the check cultivars Beaver and Agate. From this test, seed of cycle 3 was used to carry out the three cycle strain test.

The three cycle strain test involved appraising a small number of genotypes from cycle 1, 2, and 3 with a view to extrapolating a trend of selection for the resistant and susceptible populations over 3 cyles of selection. The small number of genotypes under test was due to the lack of bench space in the growth room.

The determination of the genetic factors governing the inheritance of resistance to alfalfa sickness was the object of study in the diallel strain test. Two half diallel populations were established from first cycle genotypes and the parental clones in each diallel were composed of:

Resistant Diallel - 6 X 6 Diallel cross (F1s only)

- Clone 1 (Vernal, U.S.)
- Clone 2 (Vernal, U.S.)
- Clone 3 (Beaver, Canada)
- Clone 4 (Beaver, Canada)
- Clone 5 (Vernal, U.S.)
- Clone 6 (Beaver, Canada)

Susceptible Diallel - 5 X 5 Diallel cross (F1s only)

- Clone 1 (Grimm, U.S.)
- Clone 2 (Beaver, Canada)

Clone 3 (Vernal, U.S.)
Clone 4 (Vernal, U.S.)
Clone 5 (Vernal, U.S.)

The origin of the parental clones is indicated in parenthesis.

The five field tests were concerned with the evaluation of 373 genotypes in comparison to 4 check cultivars. Of these genotypes, 156 were replicated 4 times, 109 twice, and 108 were observation rows. The genotypes included in each test depended on quantities of available seed stock. The data in the replicated tests were analyzed according to a randomized complete block design and, in some cases, the lattice design appropriate to the number of entries in each test.

3. Statistics

The data from the growth room tests and field tests were analyzed by analysis of variance (Steel and Torrie, 1960; Cochran and Cox, 1957). A split plot design (Steel and Torrie, 1960) was used to analyze the data for lattice strain test 1 and 2, and strain test 1. The main plots were the genotypes and check cultivars while subplots were years (2). The analysis of the diallels in the diallel strain test assumed preselected parents and included only F1s. Thus, Griffing's (1956) method 4 and model 1 was used.

In each growth room test, height, stand survival, and disease rating means per pot represented a replicate while for yield, the total dry matter per pot was considered a replicate. Single plant measurements were used for the diallel strain test but the means per pot were used for comparing the genotypes. For the field tests, a single row was considered a replicate. The dimensions for a row were 3.3 meters in length for lattice strain test 1 and 1.6 meters long for the other field tests. Spacing between all field rows was 45 cm.

Means in each growth room test were further analyzed by Duncan's multiple range test (Duncan, 1955) at the 5% level of probability. Field test means for strain test 2 were also analyzed by Duncan's multiple range test. For the second cycle strain test, lattice strain test 1 and 2, and strain test 1, the means of the genotypes were compared by the least significant difference (LSD) (Steel and Torrie, 1960) at the $p \leq 0.05$ and $p \leq 0.01$ levels. Means for the resistant genotypes, susceptible genotypes, and check cultivars were compared using an unpaired t-test (Steel and Torrie, 1960) in the second cycle selection test, three cycle selection test, diallel strain test, lattice strain test 1 and 2, and strain test 1. Simple correlation coefficients (r) (Steel and Torrie, 1960) between the agronomic characters were reported for each field test and growth room test. A coefficient of variability (CV) (Steel and Torrie, 1960) was calculated for each variable within each growth room test and field test.

Frequency distributions were plotted for height of the resistant, susceptible, and unselected genotypes over two cycles of selection for alfalfa sickness. The difference between the height means of cycle 2, and cycle 1 and 0 was due to a difference in sick soils used for growing the plants of cycle 2 vs cycle 0 and 1. The graphs for height in cycle 1 and 2 represent per cent of plants in each height class vs the height class. There were unequal sample sizes for the resistant, susceptible, and unselected genotypes in each cycle. No frequency distributions for disease rating in cycles 0, 1, and 2 were illustrated since there was a difference in disease rating schemes, and sick soils used between cycle 2, and cycle 0 and 1.

Narrow sense (H^2N) heritability was determined for height over 2 cycles of selection by Lush's (1940) method of parent-offspring regression where $H^2N=2b(100)$ and b=regression coefficient of open pollinated progenies on parental clones. Heritability according to Gardner (1963) was calculated in the diallel strain test for broad (H^2B) and narrow (H^2N) sense as follows:

$$H^2B= (4VGCA + 4VSCA)\ 100/\ 4VGCA + 4VSCA + VE$$

$$H^2N= (4VGCA)\ 100/\ 4VGCA + 4VSCA + VE$$

where VGCA, VSCA, and VE are the general combining ability (GCA) variance, specific combining ability (SCA) variance, and error (E) variance components, respectively. For the second cycle strain test, the broad sense heritabilities were calculated by Falconer's (1960) method of partitioning the genotypic variance. The genetic coefficient of variation

(CV) was determined by taking the square root of the genetic variance, dividing by the population mean, and multiplying by 100 (Frakes et al., 1961).

Simple linear regression equations (Steel and Torrie, 1960) were determined for the time of infection test. The equation took the form of:

$$Y' = A + B'X'$$

Y' = estimated value for Y (dependent variable)

A = Y intercept

B' = regression coefficient

X' = independent variable

The independent variable was time while height and disease rating were the dependent variables.

RESULTS

1. Pathogen

a. Soil Test 1 Some 20 soils from the vicinity of Edmonton, Alberta were assessed for the presence of alfalfa sickness and control of the pathogen. The soil sample, control treatment, and soil sample by control treatment interaction mean squares for height, yield, and stand survival were highly significant except for the control treatment mean squares in stand survival which was not significant.

Plants grown in pasteurized soil (Table 3) were significantly taller and higher yielding compared to plants from nonpasteurized soil and nonpasteurized-limed soil. Liming did not significantly increase plant height or yield in pasteurized or nonpasteurized soil.

Typical alfalfa sickness symptoms were observed on plants raised in eight of the twenty nonpasteurized soil samples. The mean height and yield of plants growing in the unpasteurized soil were 27.0 cm and 2.4 g, respectively. One particular soil sample yielded plants in nonpasteurized soil with a mean height of 13.6 cm and 0.6 g of dry matter. Plants from this soil were significantly lower in mean height and yield compared to plants from the other nonpasteurized soil samples.

b. Soil Test 2 From the analysis of variance, highly significant F-values were obtained for height, yield, and disease rating treatment mean squares. The significant mean squares for yield and disease rating were attributed to inclusion of the pasteurized sick soil treatment. The sick soil samples 1 to 5 (Table 4) from the field site grew plants which were lower in height and yield having more diseased roots than plants which were grown in pasteurized sick soil. No significant differences were found for disease rating and yield among plants grown in any of the soils sampled in the field. The plants from sick soil 1, 2, 3, and 4 were significantly taller than plants grown in sick soil 5 ($p \leq 0.05$) using Duncan's new multiple range test. Apparently, sick soil from the field site was essentially uniform in causing alfalfa sickness.

c. U.S. Soil Test Observation pots of soils from different geographic areas purported to be infected with *P. megasperma* were planted with Beaver alfalfa (Table 5). The Ontario soil produced plants with the highest disease rating, and lowest yield and height. Plants in the two Minnesota soils and California soil were taller, higher yielding and less diseased than plants from the Michigan and Ontario soils. The average disease rating of 3.29 for plants growing in these soils was considerably higher than the disease rating of plants growing in alfalfa sick soil (2.52). However, the mean yield and height of Beaver grown in sick soil was lower than the mean height and yield of plants from *P. megasperma* infected soil. External disease symptoms on alfalfa roots caused by alfalfa sickness and *P. megasperma* appeared similar. However, *P. megasperma* caused more external reddening of the alfalfa root tissue.

d. Time of Infection Test The mean squares of treatments were highly significant for height and disease rating. Plant height increased significantly (Table 6) from 2 to 7 weeks but disease ratings of the plants at 5, 6, and 7 weeks were not significantly different from each other. Figure 1 illustrates the increasing height while the disease rating has levelled off at 5 weeks. The alfalfa began to bloom at 6 weeks and was flowering at 7 weeks. Crown buds developed 6 weeks after planting.

From this time of infection test it was observed that the causal agent of alfalfa sickness primarily attacks the root tips, noncambial lateral roots, the junction where the noncambial lateral roots attach to the tap root, and the nodules, if present. On the tap root, some of the emerging lateral roots are rotted off completely and a new lateral root was observed being formed adjacent to the destroyed one. As well, some of the lateral roots, less than one-half cm from the root tip, were entirely girdled. The lateral root, in this case, did not have any evidence of lesions or browning within the area of girdling. However, on older lateral roots, the characteristic browning and lesions were observed. This suggests girdling of the lateral roots is followed by induction of lesions and browning which indicate root tissue collapse. Damirgi et al. (1977) have photographed some of these observations and presented them in their paper.

Nodules developed on about 4- to 5-week-old plants and those that were present were small, whitish and contained little, if any, leghaemoglobin. It appeared these nodules were ineffective.

Linear regression equations were calculated between disease rating and time, and height and time, and found to be highly significant. The equations were: $Y' = 1.09 + 0.31X$ and $Y'' = -6.16 + 5.96X$ where X is time, Y' is the disease rating, and Y'' is the height. Eighty-four per cent of the total variation between disease rating and time was accounted for by the first equation. In the second equation, 96% of the variation between height and time was explained.

e. Inoculation Test Pasteurized sick soil, inoculated with organisms prepared as the second bioassay, and noninoculated pasteurized sick soil were planted with different alfalfa genotypes. There were highly significant treatment mean square values detected for disease rating while mean squares of height and yield were not significant.

The plants from genotypes growing in pasteurized sick soil had clean roots, but were not significantly different in height and yield from plants growing in pasteurized inoculated sick soil (Table 7). Plants of the susceptible genotype, 1GP130, were taller, but not significantly so, in comparison to plants of the resistant genotype 2V96 in pasteurized sick soil. The resistant genotypes did not produce significantly taller, higher yielding plants in pasteurized sick soil compared to Beaver plants or plants from the susceptible genotypes. In pasteurized inoculated sick soil, the mean disease ratings of the alfalfa genotypes were not significantly different from each other. But the disease rating was significantly different for plants in pasteurized sick soil compared to plants of genotypes growing in pasteurized inoculated sick soil. Phytophthora species (Cook, 1977) were isolated from plants grown in the inoculated pasteurized sick soil.

f. Fungicide Test 1 The results from a test of two fungicides, Dexon and Benlate, are presented in Table 8. Highly significant mean squares in all three of the agronomic characters studied were obtained. Plants growing in pasteurized sick soil were taller with clean roots and higher yields than plants growing in the nonpasteurized sick soil or in sick soil treated with fungicide. The fungicide treated soil did not yield plants which were significantly different in height, disease rating or yield from plants growing in nonpasteurized sick soil. Plants from Dexon treated soil were slightly less diseased with alfalfa sickness, although not significantly, compared to plants from the nonpasteurized sick soil.

g. Fungicide Test 2 The fungicide test reported in Table 9 included Dexon, Metazoloxon, and Dowco 269 each applied to sick soil. The mean squares of the treatments were highly significant for the plant characters measured.

Mixing Dowco 269 into sick soil and mixing plus spraying resulted in plants with significantly lower disease ratings compared to plants in nonpasteurized sick soil (Table 9). The plants from Dowco 269 treated sick soil were significantly higher yielding for the soil mix than plants from unpasteurized sick soil. The Dowco 269 spray treatment was not as effective as mixing or spraying plus drench since the plants were more diseased and lower yielding when only sprayed. No significant height differences were obtained for plants from Dowco 269 treated soil compared to plants in nonpasteurized sick soil. Roots from plants in the Dowco 269 soil except the spray treatment were as white and clean as roots from plants grown in the pasteurized sick soil. There were no visual effects of Dowco 269 or Metalzoloxon on the alfalfa nodules.

Plants grown in the Metazoloxon treated sick soil had browning and lesions on the roots but the disease rating was lower compared to plants grown in nonpasteurized sick soil (Table 9). In Metazoloxon treated sick soil, plant height and yield were not significantly lower than plants grown in unpasteurized sick soil. Plants, from sick soil applied with Metazoloxon, were not significantly different in mean yield, height, or disease rating for either of the application methods.

The disease rating of 4 which was given to plants grown in Dexon treated soil was based on a small, stunted root system and very necrotic lateral roots (Table 9). No nodules were observed on any of the plant roots from Dexon treated soil. Plants grown in soil to which Dexon had been applied were lowest in yield and height, and no significant difference existed in mean height, yield, or disease rating between plants grown in Dexon soil mix or Dexon soil mix plus drench. One last result was the significant difference between plants grown in pasteurized sick soil in which they had clean roots, high yields, and were tall compared to plants from all the other fungicide treatments and the unpasteurized sick soil treatment.

h. *Strain ± Fungicide Test* The mean squares were significant for height, yield, and disease rating. The roots of the plants for the three genotypes grown in the Dowco 269 treated sick soil were rated clean and were significantly different from plants of the same genotypes growing in nonpasteurized sick soil (Table 10). Plants of the genotype 1V12 yielded significantly more in Dowco 269 sick soil compared to nonpasteurized sick soil. There was no significant difference between the mean height of plants of each genotype grown in Dowco 269 sick soil compared to nonpasteurized sick soil. The plants from each genotype were significantly higher in mean height, and yield, and lower in mean disease rating when grown in pasteurized sick soil vs nonpasteurized sick soil. No significant differences were detected between 1V12 plants grown in either pasteurized sick soil or Dowco 269 sick soil for height, yield, or disease rating. However, for the other 2 genotypes the plants were taller and higher yielding when grown in pasteurized sick soil as compared to the Dowco 269 sick soil.

The plants of 1GP130 grown in pasteurized sick soil illustrated that the susceptible genotype could yield as much as plants of the resistant genotype 1V12 (Table 10). However, when plants of 1V12 and 1GP130 were grown in nonpasteurized sick soil, the means of plants for the resistant genotype were significantly different in height (27%), disease rating (-22%), and yield (35%) compared to the mean of plants from the susceptible genotype. The means of the resistant genotypes were also significantly greater in height (15%) and disease rating (-23%) relative to the mean of Beaver plants.

2. Plant

a. Second Cycle Selection Test Highly significant genotype mean squares for height, yield, and disease rating were found. The mean for plants from the resistant genotypes was greater by 11% for height, 18% for yield, and 10% less for disease rating compared to the mean of plants from the susceptible genotypes (Table 11). These mean diffences were significant at the 1% probability level for height and disease rating whereas the yield difference was significant at the 5% probability level. The mean of the resistant genotypes was 11% higher than the mean of the Beaver genotype and this difference was significant at $p \leq 0.05$. The range of mean yields and disease ratings was broader for plants from the resistant genotypes as compared to plants from the susceptible genotypes.

The genotype, 2V96, was the most outstanding since this genotype had the highest yielding plants with a low disease rating (Appendix 3). The susceptible genotype, 2VP60, had the shortest, lowest yielding plants while plants of the resistant genotype, 2V44A, were the lowest in disease rating. The cultivars Beaver and Agate, planted in nonpasteurized sick soil, did not perform as well as a number of the resistant genotypes (2V96, 2V102, 2V12) in yield and disease rating. Beaver plants grown in pasteurized sick soil outperformed all of the genotypes in height, yield, and disease rating.

b. Three Cycle Strain Test A test of lines resistant and susceptible to alfalfa sickness from 3 cycles of selection is presented in Table 12. Highly significant genotype mean squares were found for height, yield, and disease rating. The mean of all resistant genoytpes indicated the plants were significantly taller, less diseased, and yielded more in comparison to the mean of plants from all the susceptible genotypes. These significant mean differences ($p \leq 0.05$) were 39% (height), -14% (disease rating), and 80% (yield).

In the resistant genotypes, the mean of plants from cycles 1, 2, and 3 were not significantly different from each other in height, disease rating, and yield (Table 12). Means of cycle 2 and 3 in the resistant genotypes were significantly different ($p \leq 0.05$) in height from the means of plants in cycle 1 and 3 of the susceptible genotypes. Genotypes 1V12 and 2V102 produced plants which were significantly taller (11%), less diseased (-22%), and higher yielding (160%) than plants from 2GP130 and 3GP130. The range of mean heights, yields, and disease rating was greater for plants of the resistant genotypes compared to plants of the susceptible genotypes. Beaver grown in the pasteurized sick soil, produced the highest yielding, tallest plants with clean roots.

c. Diallel Strain Test Progenies from a six by six diallel of resistant genotypes, and susceptible genotypes from a five by five diallel were tested and the results presented in Table 13. The mean squares were highly significant for the genotypes in height, yield, and disease rating. Mean disease rating (-26%), yield (107%), and height (38%) of plants in the resistant diallel were significantly different ($p \leq 0.01$) compared to plants from the susceptible diallel.

Plants of the crosses 1V12 by 1B179, 1B179 by 1V9, and 1B152 by 1V9 were taller, higher yielding and had a lower average disease rating (2.22) in comparison to Beaver plants and other resistant and susceptible crosses (Table 13). The resistant crosses 1V57 by 1B152 and 1V57 by 1B103 had shorter and more diseased plants than plants from the rest of the resistant crosses. Plants of two genotypes, a second cycle (2V96) and a third cycle (3V102C), were significantly different from each other in disease rating.

Within the susceptible diallel, plants of the crosses 1GP130 by 1VP17 and 1BP113 by 1VP62 were the highest yielding and least diseased (Table 13). The cross in the susceptible diallel which had the shortest, lowest yielding plants and a poor disease rating was 1VP17 by 1VP58. Plants of the susceptible genotype, 1GP130, were less diseased, taller, and produced more yield compared to the mean disease rating, height, and yield of plants from the susceptible diallel. Beaver alfalfa grown in the pasteurized sick soil had clean roots and the highest yield.

Mean squares calculated in the analysis of height and disease rating for the resistant and susceptible diallels are given in Table 14. The highly significant general combining ability (GCA) mean square is larger than the highly significant specific combining ability (SCA) mean square in the resistant diallel for height and disease rating. In the susceptible diallel, the significant SCA mean square is equal to the GCA mean square for the character height and larger than GCA for disease rating.

The estimates of combining ability (Table 15) show that genotypes 1B179 and 1V9 in the resistant diallel would be good combiners as parents based on the estimates of GCA effects. If 1V57 were used as a parent, it would be a poor general combiner in a multiclone synthetic since short, diseased plants would be produced. In the susceptible diallel, short plants with diseased roots would be obtained by making crosses with 1VP58 as a parent. However, crosses with 1GP130 as a parent in a synthetic of many clones would be desirable since this good general combiner would propagate tall plants with little disease. The susceptible cross 1VP17 by 1VP58 would be a suitable 2-clone hybrid according to the estimates of SCA effects as short, diseased plants would result. The cross 1V12 by 1V57 should be a good specific combination due to a high positive SCA effect for height and high negative SCA effect for disease rating.

A comparison of the resistant and susceptible genotypes averaged over 3 cycles of selection with unselected Beaver is presented in Table 16. The resistant genotype mean was 9% higher than the Beaver genotype mean and 26% higher than the susceptible genotype mean in height. For yield, the mean of the resistant genotypes was 19% and 54% greater than the Beaver and susceptible genotype means, respectively. In disease rating, the resistant genotype mean was 8% and 16% less than the Beaver and susceptible genotype means, respectively.

d. Lattice Strain Test 1 The 1976 and 1977 data indicated highly significant differences existed among individual genotypes in mean heights, yields, and performance ratings. The mean squares of the individual genotypes for the lattice design was highly significant but the lattice design did not provide any gain in accuracy for the characters analyzed over the randomized complete block design. Thus, the randomized complete block design was used in evaluating the results. Means of the genotypes averaged over 2 cycles of selection are presented in Table 17 for the characters measured in 1976 and 1977. The range of means in the resistant genotypes for the agronomic characters studied was wider than either the range of means in the susceptible or cultivar genotypes. The genotype mean of the resistant plants was higher in yield, height, and performance rating, but not significantly so, compared to the mean of plants from either susceptible genotypes or cultivar genotypes.

The mean of plants from 2R163 and 1B29 was significantly greater than the mean of plants from 1GP130 and 1VP17 in height (1976-11%), yield (1976-77%, 1977-70%), and performance rating (35%) (Appendix 4). The mean of plants from these genotypes (2B66A, 1B24, 1B29, 2R163, 2B106, 1V57, 1B73, 2B76, 1B110, 2V13) was higher than the mean of the susceptible genotypes by 6%, 4%, 34%, 28%, and 20% in height 1976, 1977; yield 1976, 1977; and performance rating, respectively. Compared to the mean of plants from the cultivar genotypes, these differences were even greater except for yield in 1977.

e. Lattice Strain Test 2 When analyzed as a randomized complete block design, highly significant individual genotype mean squares were found for height in 1976 and 1977, and yield in 1977. The rectangular lattice design increased accuracy over the randomized complete block design (100%) by 145% and 114% for height in 1976 and 1977, respectively. Thus, height means have been adjusted according to the lattice design analysis. The lattice design was not any more effective than the randomized complete block design for yield in 1977 and the 1977 yield means are presented as being analyzed by the randomized complete block design. The means of plants from resistant, susceptible, and cultivar genotypes averaged over 2 cycles of selection were not significantly different from each other in height for 1976 and 1977 and in yield (Table 18).

The mean of plants from the following resistant genotypes (Appendix 5) - 2V8, 1G20, 2V99A, 2V62A, 2G158 - was 10% and 9% higher for height in 1976 and 1977, and 54% greater for yield compared to the mean of plants from the susceptible genotypes (2VP54, 1VP58, 2VP43, 1GP133, 1GP21). In contrast to the mean of the cultivar plants, the resistant genotype mean of those plants was 4%, 3%, and 9% higher for 1976 height, 1977 height, and yield respectively. Plants from the genotype, 1G20, were significantly higher than 1GP21 plants in height (1976-33%, 1977-15%), and yield (76%).

f. Strain Test 1 The randomized complete block design analysis for height in 1976 and 1977 and yield in 1977 indicated significant mean squares for individual genotypes. The lattice design did not improve the accuracy of the genotype mean squares over the randomized complete block design for any of the agronomic characters studied. Thus, results from the data were presented as being analyzed by the randomized complete block design. No significant differences were detected among the means of plants from resistant, susceptible, or cultivar genotypes (Table 19).

The mean of plants from the resistant genotypes (2V56A, 2V51, 2B75A, 2B110, 2B74, 2B108A) was above the mean of plants from the susceptible genotypes 1VP51, 1VP60, 1VP19, 1GP20, 1RP1, and 1RP62 by 22% and 20% in height for 1976 and 1977, respectively, and 130% for yield in 1977 (Appendix 6). For these same resistant genotypes, their mean was larger than the mean of plants from the cultivar genotypes by 4% and 5% in height for 1976 and 1977 and 45% in yield. The genotype, 2V56A, was a superior line since its plants were tall and high yielding. In contrast, plants from 1VP19 were short and low yielding.

g. Strain Test 2 Highly significant mean squares for individual genotypes were obtained for the 2 characters measured. The mean of plants from resistant genotypes (Table 20) averaged over 2 cycles of selection was significantly less diseased (3%) than the mean of plants from either the susceptible genotypes or cultivar genotypes. The range of mean heights and disease ratings was widest for plants from the resistant genotypes, next the susceptible genotypes, and last, the cultivar genotypes. Plants of the resistant genotype 2V96 were significantly taller by 18% and had 13% less diseased roots than plants of the susceptible genotype 2VP98. The mean disease rating of Beaver plants in strain test 2 (2.73) (Table 20) was comparable to the growth room tests in which Beaver plants were rated 2.71 (Table 16).

Initial field infection in strain test 2 was observed to occur a month-and-a-half after seeding at which time the plants were 10 cm tall and the roots 8 to 10 cm long. There was infection on the root tips and lateral noncambial roots. Observation of plant roots to a depth of 15 to 20 cm in strain test 2 following the overwintering period revealed the lateral noncambial roots to be completely rotted although no lesions were observed on the tap root. It was also observed in the area adjacent to the field site that alfalfa was more severely affected by alfalfa sickness than check cultivars in the field tests. The alfalfa in the area adjoining the field site had been undersown with barley and no control of weeds was practiced in the establishment year and second year.

h. Observational Strain Test This unreplicated field test included plants with a wider range of heights in the establishment year than in 1976 (Table 21). Consequently, the standard deviation was greater for height in 1977. The range of heights and yields for 1976 and 1977 were the widest of any of the field tests. The mean height and yield of plants for the observational strain test were the highest of the 1.6 m long field tests which included lattice strain test 2, strain test 1 and 2.

Observations recorded were consistent for 1976 and 1977 and were concerned with the performance of the check cultivars. Vernal produced plants with a mean height and yield which was higher than the means of the other check cultivars in lattice strain test 1 (Appendix 4) while the mean of Roamer plants was low in height and yield in lattice strain test 1 and strain test 1 (Appendices 4 and 6). The mean of Beaver plants in lattice strain test 2 and strain test 1 were the tallest and highest yielding (Appendices 5 and 6).

The summary of the analysis of the field tests for 1976 and 1977 (Table 21) illustrates that lattice strain test 1 had plants with the highest mean height in 1976 and 1977 followed by the observational strain test, lattice strain test 2 and strain test 1. The highest standard deviation for height was in lattice strain test 2 while the lowest standard deviation for height was in lattice strain test 2. The 1977 mean yield, coefficient of variability, standard deviation, and range of means for plants in lattice strain test 2 and strain test 1 were very similar. The measurement of plant yield and height was more accurate in 1977 than in 1976 according to the coefficients of variability for the field tests.

The split plot analysis of plant height for lattice strain test 1 and 2, and strain test 1, and of plant yield for lattice strain test 1 for 1976 and 1977 indicated significant genotype mean squares. The year by replication, and year by genotype interaction mean squares were highly significant except the year by replication interaction mean square for yield in lattice strain test 2 which was only significant. Data from the split plot analyses were not presented since the coefficients of variability did not indicate any gain in accuracy: for yield in lattice strain test 1 and height in lattice strain test 2; or very little, for height in lattice strain test 1 and strain test 1 (Table 21).

In the analysis of the four cultivars from lattice strain 1 and 2, there were no significant mean squares for the field test by cultivar interaction for height in 1976 and 1977, and yield in 1977. There were significant F-values for field test mean squares for height (1976) and yield (1977). When these cultivars were analyzed over years within each field test, there was significant mean squares for year by cultivar interaction for height in lattice strain test 1 and 2, and strain test 1, and for yield in lattice strain test 1.

No post-emergence damping-off was observed in any of the field tests or growth room tests. As well, no record of death in which the death could be attributed to alfalfa sickness for any of the growth room tests or field tests was noted. It was observed that horsetail (Equistem arvense) was prevalent in the field site and adjoining field.

i. Correlations Simple correlation coefficients among agronomic characters utilized in studying the alfalfa sickness organism were highly significant (Table 22). The only exceptions were for the correlations between stand survival and yield (r=0.21*), and stand survival and height (r=0.16 n.s.) in soil test 1; yield and disease rating in the inoculation test (Table 22); and height and disease rating in the inoculation test and strain + fungicide test (Table 22).

In soil test 1, the small variation in stand survival did not result in a highly significant correlation between it and yield or height. The correlation between height and disease rating (r=0.95**) was positive in the time of infection test since disease rating increased from 1.54 to 2.99 as did the height (4.8 to 33.4 cm) (Table 22). The nonsignificant r-values in the inoculation test and the low r-value (r=0.34*) in the strain + fungicide test were a result of the small variation in disease rating (Table 22). A positive correlation between yield and disease rating in the inoculation test was due to higher yielding plants growing in the inoculated pasteurzied sick soil (Table 22).

Correlation coefficients among agronomic characters used to assess the control of the pathogen by plant breeding are presented in Table 23 and 24. The r-values in the growth room tests (Table 23) were highly significant. The correlation between height and disease rating for the second cycle strain test was the lowest of the three growth room tests (Table 23). The correlation coefficients between the field tests and growth tests for yield and height were low and nonsignificant (r=-0.18 to 0.12). Disease rating in the growth room and performance rating in the field tests were not significantly correlated (r=-0.11 to -0.04).

In the field tests, all the correlations were highly significant with the exception of disease rating vs height in 1976 for strain test 2 which was nonsignificant (Table 24). The correlations between the variables measured in different years was lower than the correlations between variables studied within each year. The simple correlation coefficients in the observational strain test were 0.43** between height in 1976 and 1977, 0.36** for height (1976) and yield (1977), and 0.47** for height (1977) and yield (1977).

j. Heritability The analysis of height over 2 cycles of selection for resistance and susceptibility to alfalfa sickness in the growth room is illustrated in Figures 2, 3, and 4. Figure 2 depicts the response of unselected material in sick soil and in nonsick soil. The difference between the mean heights of plants in sick soil (12.2) and nonsick soil (18.0) was significant at the 1% level of probability. In Figure 3, the frequency distribution of the resistant genotypes and susceptible genotypes is within the frequency distribution of the unselected material. The frequency distribution of the resistant genotypes in Figure 4 is progressing upwards and part of this distribution is outside the frequency distribution of the unselected material.

The resistant genotype mean height (cycle 1-18.1 cm;cycle 2-26.5 cm) was not significantly different from the unselected genotype mean height (cycle 1-18.0 cm; cycle 2-23.9 cm) in cycle 1 but was in cycle 2 at $p \leq 0.01$ (Table 25). The mean height of the resistant genotypes was significantly different from the mean height of the susceptible genotypes at p≤0.01 in cycless 1 and 2. This difference amounted to 15% and 10% for height in cycle 1 and 2, respectively. The susceptible and resistant genotype means were not significantly different in disease rating for cycle 1 but in cycle 2, the resistant genotype mean was 10% lower (p≤0.01) in disease rating than the susceptible genotype mean. Selection intensity was 13% in cycle 0 while for cycles 1 and 2, the intensity increased to 6% and 8%, respectively, for the resistant genotypes. The narrow sense heritability of height for the combined analysis of the resistant and susceptible genotypes was low decreasing from 30% in cycle 1 to 18% in cycle 2 for height (Table 26). The regression coefficients used in estimating heritability were nonsignificant for height in the resistant and susceptible genotypes analyzed separately (Table 26).

The narrow sense heritability for disease rating was in direct contrast to the heritability of height as it increased from 12% to 22% in cycles 1 to 2 for the resistant plus susceptible genotypes combined (Table 26). Heritability of disease rating in the susceptible genotype increased from 12% to 18% in cycle 1 to cycle 2 (Table 26). However, the resistant genotype regression coefficients for disease rating were nonsignificant in cycles 1 and 2 (Table 26).

The values of broad sense heritability from the second cycle selection test were 42% and 4% for height and disease rating in the susceptible genotypes. The resistant genotype heritability estimate was 41% and 51% for height and disease rating. Height was highly heritable in the susceptible and resistant genotypes but the heritabiblity of disease rating in the susceptible genotypes was low. The genetic coefficient of variability was highest for disease rating (8.3%) in the resistant genotypes and lowest for disease rating (1.3%) in the susceptible genotypes. The genetic CV was intermediate for height at 7.2% and 6.9% for the resistant and susceptible genotypes, respectively.

The estimates of heritability for height in the diallel strain test (Table 14) ranged from 98% to 99% for broad sense heritability and 32% to 46% for narrow sense heritability. Heritability of disease rating was high for broad sense at 96% to 98% and lower for narrow sense at 11% to 33%. Higher heritabilities were obtained for the resistant diallel relative to the susceptible diallel. These differences were significant at $p \leq 0.01$ for broad and narrow sense heritability of disease rating and for narrow sense heritability of height. The genetic coefficient of variability was lowest for disease rating in the susceptible diallel.

DISCUSSION

1. Pathogen

a. Interaction of Plant and Pathogen Infection of alfalfa seedlings by the alfalfa sickness pathogen did not increase after 5 weeks (Table 6; Figure 1). The disease infection reached a plateau which may have been caused by either the inoculum level in the substrate, or, the plants developing a resistance to the disease. This situation coincides with observations that infected fields tend to maintain good stands but remain unproductive (Bolton, 1977). Therefore, the optimum time to rate alfalfa sickness symptoms is at 5 weeks under the growth room conditions maintained in this test.

Highly significant correlation coefficients among disease rating, yield, and height (Table 22) indicated a close association among the 3 variables. No post-emergence damping-off or record of death of plants due to alfalfa sickness occurred in the growth room tests or field tests. These data indicate a relationship between disease infection and reduction in height and yield of plants grown in sick soil.

The disease rating in the field, for strain test 2, took place two-and-a-half months after seeding. It should have been carried out half-a-month previously so that ratings in the field would have taken place at the same growth stage of the plants as that in the growth room. However, the mean disease rating of Beaver in the growth room (Table 16) and in the field (Table 20) were similar. But the correlations between agronomic characters of genotypes evaluated in the growth room tests and field tests were low and nonsignificant. This would indicate the need for field testing of genotypes.

To test the hypothesis that alfalfa sickness is identical to Phytophthora root rot caused by P. megasperma, soils from areas in the United States and Ottawa, Ontario, reputed to be infected with the pathogen, were sown with alfalfa (Table 5). The disease ratings of plants grown on these soils were considerably greater as compared to the ratings of plants grown on alfalfa sick soil. On the assumption P. megasperma causes alfalfa sickness, this would imply either a lower inoculum level is present in soils from central Alberta than from the four Phytophthora-infected soils, or, P. megasperma was more virulent than it was in the alfalfa sick soils. A visual examination of plant roots from the Phytophthora-infected soil and sick soil revealed both diseases produced the same external symptoms. There was not much difference in mean yield and height of the plants grown in sick soil relative to the mean of plants raised in the Phytophthora-infected soils. The similarity of disease symptoms caused by each pathogen suggests that P. megasperma induces alfalfa sickness.

Examining the environmental conditions necessary for P. megasperma to survive, the fungus has been found to be endemic to poorly drained soils or soils with high water tables (Frosheiser, 1968). These conditions may not exist to in those parts of Canada where alfalfa sickness is found. The influence of soil moisture on the severity of alfalfa sickness has not been studied and should be investigated. Furthermore, soils in poorly drained areas usually have a high clay content. The texture of the soil at the field location (Spruce Grove, Alberta) plus other alfalfa sick areas (Bolton, 1977) suggests alfalfa sickness occurs on light-textured soils.

In relation to the field location at Spruce Grove, Alberta, the occurrence of _Equisetum arvense_ or horsetail would appear to indicate the soil has a high water table. The internal drainage and northward sloping topography of the field location may also contribute to the height of the water table. Frosheiser (1969) found that 17 cm of rain fell in the month of June where _P. megasperma_ was isolated. Almost twice that amount of rain fell in the 4-month intervals from 1969 to 1976 at the field site (Appendix 2). These observations suggest environmental conditions favorable for survival of _P. megasperma_ .

In the inoculation test, isolates of organisms from alfalfa sick soil, and cultures of _P. megasperma_ , when applied to pasteurized sick soil, caused small lesions on alfalfa roots (Table 7). The plants were rated 2 and no significant differences were detected among plants of the different genotypes growing in inoculated pasteurized sick soil. The failure to induce severe symptoms of alfalfa sickness or Phytophthora root rot implies that either the isolates and cultures were too old to be virulent, or, the plants were developing a resistance to the disease. This experiment should be repeated to determine if this was indeed the case. The inoculation technique, if successful, could provide a reliable method of infection for future studies, and so improve the accuracy with which genotypes for resistance could be made.

Plants from the cultivar Agate, in the second cycle strain test, proved to be significantly more diseased than Beaver plants (Appendix 3). In yield and height, the Agate plants were not significantly different from the Beaver plants (Appendix 3). Agate is reputed to be winterhardy and resistant to _P. megasperma_ (Lueschen _et al._, 1976), but it was not bred for western Canadian conditions and may not be adapted to this area. In strain test 1 (Appendix 6), the results showed Beaver plants to be significantly taller than Agate by 30% and 18% in 1976 and 1977. Thus, it was concluded that Agate was at best equal to Beaver. This would indicate Agate is not resistant to alfalfa sickness and could suggest alfalfa sickness is not caused by _P. megasperma_ .

Other research directly related to the interaction of the pathogen and plant is concerned with determining the extent and distribution of alfalfa sickness in Alberta (Cook, 1977). Two other areas which should be pursued are; first, a study of the effects of long term storage on the sickness pathogen in the soil, and its ability to reinfect alfalfa, and secondly, a rotation study is needed to determine the effect of the pathogen on successive stands of alfalfa grown in sick soil. These studies could be undertaken in a growth room.

In summary, the following conclusions were formulated from our knowledge of the interaction of the pathogen and plant. Alfalfa sickness infection is initiated in alfalfa seedlings, a month to a month-and-a-half old, at the root tips, lateral noncambial roots, and the nodules, when present. It is suggested that a low or nonpathogenic level of inoculum exists in certain soils and during the life of the first stand this level increases, and persists in the soil. In succeeding alfalfa stands, the abundance of inoculum present ensures a parasitic association between the plant and pathogen. The plant's growth is severely restricted for the life of the stand and alfalfa yields are low. The nodules also may become ineffective so that the nitrogen metabolism of the plant may become severely affected resulting in stunting of the plant and yellowing of the leaves. This would support the observations made by Goettel (1962) and Webster *et al.* (1967) that after the first stand of alfalfa, subsequent stands are affected by alfalfa sickness, and show typical symptoms.

b. *Control by Physical and Chemical Treatments* The first control measure tested was heat and the results from this physical treatment confirmed earlier observations that the agent causing alfalfa sickness was biological (Webster et al., 1967). Pasteurizing sick soil either substantially reduced alfalfa sickness or eradicated it. Soil treated in this manner always produced tall plants with high yields and clean roots (Tables 3, 4, 8, 9, and 10). This agrees with the evidence presented by Webster et al. (1967) who used the soil sterilant, Vapam, and steam sterilization of sick soil, and Goettel (1962) who noted sterilizing sick soil improved yields of plants growing in sick soil. The pasteurization of alfalfa sick soils on a field scale would be impractical and economically not feasible.

Other physical treatments such as liming, fertilizing, and soil moisture, applied at optimum levels, did not increase yield, height, or lower disease rating of plants growing in sick soil to a level approaching that of plants growing in pasteurized sick soil (Tables 3, 4, 8, 9, and 10). Webster et al. (1967) and McElgunn and Heinrichs (1970) also reached the same conclusions in their studies using fertilizer, lime, and moisture, or any combination of these factors to correct growth of alfalfa in sick soil.

The second type of control treatment tested was chemical fungicides. Dexon did not control alfalfa sickness as the roots of plants from sick soil treated with this fungicide were as diseased as roots from plants in the nonpasteurized sick soil (Tables 8 and 9). This treatment did not result in taller, higher yielding plants. The application rate of Dexon was believed to be phytotoxic when applied at 10 times the recommended rate in fungicide test 2 (Table 9). Benlate is purported to control organisms other than pythiaceous fungi, and its use might be expected to result in more severe root symptoms, lower plant yield and plant height. This was not the case (Table 8). Plants from the Benlate treated sick soil were not significantly different for height, yield, and disease rating from alfalfa grown in the Dexon treated sick soil or in nonpasteurized sick soil.

Metazoloxon provided some protection as the disease rating of alfalfa roots was less for this treatment than for roots of plants grown in nonpasteurized sick soil (Table 9). A higher yield and height of plants grown in Metazoloxon treated sick soil compared to plants from unpasteurized sick soil was expected. The results did not show this.

Dowco 269 was the most promising fungicide (Table 9). Roots of plants from Dowco 269 treated sick soil were rated clean and healthy. The yield of this fungicide treated sick soil was significantly greater than the yield of plants from nonpasteurized sick soil. Height was not significantly different. The application rate may require further study and some adjustment for optimum growth of alfalfa in Dowco 269 treated sick soil.

The drench-mix application procedure did not improve height or yield, or decrease disease rating of the alfalfa plants compared to plants from the soil mix (Table 9). This was true for Dexon and Metazoloxon. Spraying Dowco 269 controlled alfalfa sickness but not as effectively as Dowco 269 soil mix or spray-soil mix applications judged by the height, yield, and disease rating of plants from these treatments.

Stuteville (1976) stated Dowco 269 is active against soil-borne pythiaceous fungi. The fungicide gave seedling protection to alfalfa growing in soils infested with P. megasperma (Stuteville, 1976). Results from our experiments for Dowco 269 indicated alfalfa seedlings were protected when grown in alfalfa sick soil (Tables 9 and 10). This would imply that alfalfa sickness is a pythiaceous fungus.

Beaver plants grown in Dowco 269 treated sick soil were significantly less diseased and higher yielding than Beaver plants in nonpasteurized sick soil (Table 10). The plants of the resistant genotype (1V12) were not significantly different in height, yield, or disease rating when grown in either Dowco 269 treated sick soil or pasteurized sick soil. When 1V12 plants were grown in sick soil with and without Dowco 269, there was a significant difference between the mean disease ratings. Beaver plants were shorter in height, although not significantly so, than plants of 1V12 in Dowco 269 treated sick soil. This suggested that chemicals and plant selection, in combination, should be further investigated.

Dowco 269 controlled alfalfa sickness by either drenching it on the soil surface (Table 10), or mixing it in with the sick soil (Table 9). Both methods of application would seem practical, but the cost of the chemical may be a limiting factor for commercial purposes. Unfortunately, Dowco 269 has been withdrawn (Evans, 1976) for further evaluation, but, these positive results indicate that fungicidal control is possible.

In pasteurized sick soil, the plants of a resistant genotype, susceptible genotype, and Beaver were not significantly different from each other in height, yield, or disease rating (Table 10). There was a significant difference between each genotype in pasteurized sick soil vs nonpasteurized sick soil for the above agronomic characters. However, when plants of the genotypes were grown in nonpasteurized sick soil, the resistant genotype was significantly better than either the suceptible genotype or Beaver in height and disease rating, and significantly greater than the susceptible genotype in yield. These results indicate that plant breeding is a suitable alternative to chemical applications.

2. Plant

a. Control by Plant Breeding In very few instances were plants of the resistant genotypes, grown in nonpasteurized sick soil, taller, heavier, or less diseased than Beaver plants grown in pasteurized sick soil (Tables 11, 12, 13, and 16). When all the genotypes were grown in sick soil, those selected for resistance yielded significantly taller, heavier plants with less disease than either plants from susceptible genotypes or from Beaver (Tables 11, 12, 13, and 16). Evidently, there was a certain amount of misclassification when the cycle 0 plants were selected for resistance and susceptibility. It is also possible some plants may have escaped infection by alfalfa sickness. The results of these 2 possibilities are reflected in the field tests where some susceptible genotypes were considerably taller and higher yielding than certain resistant genotypes or cultivars (Appendices 4, 5, and 6). In the field tests, the mean of plants from the resistant genotypes was not significantly greater than the mean of susceptible genotypes or cultivar genotypes for height, yield, performance rating, or disease rating (Tables 17, 18, 19, and 20).

The second cycle strain test, three cycle strain test, and the field tests, all showed a wider range of agronomic characters for the resistant genotypes as compared to the susceptible genotypes when averaged over cycles of selection (Tables 11, 12, 17, 18, 19, and 20). The presence of a greater number of different gene combinations when expressed phenotypically would account for the wider range of those agronomic characters studied within the resistant genotypes as compared to the susceptible genotypes.

Resistant and susceptible genotypes, grown in pasteurized sick soil, were not significantly different in height and yield (Tables 7 and 10), but in nonpasteurized sick soil, there were significant differences in height, yield, and disease rating. These results are similar to the height response of unselected genotypes grown in sick and nonsick soils (Figure 2).

Cycle 1 resistant genotypes were significantly different in mean height from the susceptible genotypes (Table 25; Figure 3). In cycle 2, the resistant genotype was significantly different from the plants of the susceptible genotypes in height and disease rating, and from the plants of the unselected genotypes in height (Table 25; Figure 4). These results suggest that selection for alfalfa sickness resistance is being controlled by many genes and consequently, change in response to selection is slow. The selection responses of the resistant population and the susceptible population follow the trend illustrated by Sheldon (1963), Robertson and Reeve (1952), and Woodworth _et al._ (1952). It would be interesting to evaluate three more cycles of selection to determine if a major divergence occurs between the two populations. It was concluded directional selection for resistance and susceptibility to alfalfa sickness was effective in the growth room, and should be continued.

In contrast to the above results, data from the three cycle strain test (Table 12) indicated a downward trend from cycle 2 to 3 in yield, height, and disease rating for the resistant genotypes. The differences from cycle 2 to 3 were not significant and this would suggest selection was not effective. However, the sample of genotypes representing each cycle was small. Also, in Figures 3 and 4 for cycle 1 and 2, respectively, the data showed positive responses to selection.

Correlations between height vs disease rating and yield vs disease rating in the growth room tests involving the plant were high and significant (Table 23). The assumption was made that taller, higher yielding plants would be resistant to disease, while shorter plants with lower yields would be susceptible. The field tests were interpreted with this assumption in mind.

The lattice design for analyzing the field tests gave no improvement in accuracy over the randomized complete block design with the exception of lattice strain test 2. The effectiveness of the lattice design for lattice strain test 2 in 1976 was due to differential weeding. Thus most of the variation within each field test ran the length of the field test or in a north-south direction.

There were differences among the means of the four cultivars from each field test for height and yield in 1976 and 1977. It appears that a genotype by year interaction was responsible for these differences. However, there was no field test by cultivar interaction for height (1977) and yield (1977). This supports data from soil test 2 (Table 4) and confirms that the field site was uniformly sick throughout.

Establishment-year results were less reliable than the second-year results as the coefficients of variability were higher for 1976 than for 1977 (Table 21). Genotypes which performed consistently in the field and in the growth room included 2V96, 2V15, 2VP42, 1GP130, and 1RP188. Some lines such as 2V12, 2B29, and 2BP113 were inconsistent in their performance. Plants from the genotype, 2V12, were highly resistant in the growth room (Table 11) but the height, yield, and performance rating of its plants did not reveal it to be a superior genotype in the field (Appendix 4). The genotypes, 2B29 and 2BP113 were just the opposite, doing poorly in the growth room (Table 11) but performing well in the field (Appendix 4).

With data from the growth room tests and field tests, a multiclone synthetic cultivar resistant to alfalfa sickness could be developed. The synthetic would include the genotypes 1B29, 1B73, 2B76, 2V13, 2V96, 2R163, and 2R187. A synthetic susceptible to alfalfa sickness would consist of clones from the following genotypes, 1GP20, 1GP21, 1GP130, 1RP1, 1RP188, and 1VP19. The synthetics could be increased for seed and tested at different locations in field trails to study their agronomic merit.

A nitrogen fixation experiment conducted in the growth room by Tan (1977) compared 20 commercial cultivars plus 5 selected lines. Of the 5 selected lines, 2 were resistant to alfalfa sickness (R2, R3). The results indicated that when the different genotypes were inoculated with commercial inoculum, plants from 2V96 (R2) were significantly higher yielding than the other entries and were 36% above the experimental mean. The total seedling dry weight of 2V12

(R3) plants was about 106% more than the experimental mean. Plants of 2V12 (R2) had a higher yield of nitrogen (30% above the average) while 2V96 (R3) plants were 6% below the overall mean. Thus, some genotypes which were resistant to alfalfa sickness appear to be inherently high yielders of dry matter and nitrogen.

Results from the growth room tests suggest that the genetic factor governing the reaction to alfalfa sickness is controlled by many genes. This conclusion is based on the high broad sense heritabilities in the diallel strain test (Table 14) and second cycle strain test (Table 11), and the slow progress in selecting for resistance and susceptibility in cycle 1 and 2 (Figure 3 and 4).

The high broad sense heritabilities in the resistant and susceptible diallels imply a high amount of epistatic, intraallelic, and interallelic interactions (Table 14). As a result, the high broad sense heritabilities for disease rating and height of the resistant and susceptible diallels would support the suggestion that nonadditive genetic variation has been exhausted. Furthermore, the low narrow sense heritabilities for height and disease rating of the resistant and susceptible diallels indicates that additive genetic variation was small. These data agree with those reported by Adams and Semeniuk (1958) who stated that in one generation, additive genetic variation could be depleted in selecting for leafspot resistance.

Genetic variability was influenced by additive and nonadditive gene action in the resistant and susceptible diallels, respectively, since the ratio of GCA to SCA was small or equal (Table 14). These results are expected since the parents of the progeny in the diallels have been subject to one cycle of selection to alfalfa sickness. The results agree with those of Singh and Lesins (1971) who found SCA to more important than GCA since the clones had been selected for GCA. Two-clone synthetics in the susceptible diallel could be selected on the basis of superior SCA due to SCA being equal to or larger than GCA. In contrast, the resistant diallel indicated that additive genetic variation was large and GCA greater than SCA. Multiclone synthetics could be formed in the resistant diallel based on the genetic differences of the parental clones.

Disease resistance and susceptibility were highly heritable in the diallels (Table 14) and this agrees with Twamely's (1974) report. The heritability of resistance was higher than that of susceptibility. However, with additive genetic variation being low, and alfalfa sickness being polygenically inherited, a number of cycles of selection would be necessary to fix favorable combinations of genes to provide a high degree of resistance and susceptibility to alfalfa sickness.

Broad sense heritability estimates calculated in the second cycle strain test supported the data obtained in the diallel strain test (Table 14). In the second cycle strain test, the heritability estimates were high ranging from 41%

to 51% for height and disease rating in the resistant and susceptible genotypes except for disease rating in the susceptible diallel (4%). The genetic CV was low for disease rating in the susceptible genotypes indicating that genotypic variation was being depleted. The genetic CV was also low for disease rating in the susceptible diallel (Table 14).

Response over 2 cycles of selection for alfalfa sickness resistance has led to increased height and lower disease ratings (Table 26). Low narrow sense heritability estimates for height and disease rating in cycle 1 and 2 would seem to indicate additive genetic variation was small. These estimates are consistent with the results obtained in the diallel strain test (Table 14). Thus, breeding for resistance and susceptibility to alfalfa sickness must be based on consecutive cycles of recurrent selection involving well-replicated progeny tests.

Three methods of calculating heritability were used, two relying on analysis of variance, and the other on regression. A more accurate analysis was suggested to account for the difference between the broad sense heritability estimates obtained in the diallel strain test (Table 14) as compared to the second cycle strain test (Table 26). The analysis of variance in the diallel strain test was based on single plants while in the second cycle strain test, the mean of plants per pot was used. The narrow sense heritability estimates from the regression of offspring on parent (Table 26) agreed with the estimates in the diallels (Table 14) which confirms results from Swanson et al. (1974).

Busbice et al. (1972) tabulated the possible range of segregates after 1, 2, and 3 generations of random mating. The assumption in determining the array of genotypes is that the original cross is between two tetragenic lines. In light of these calculations, we have evaluated 2 cycles of selection with random mating and could expect the duplex, triallelic, and tetragenic genotypes to be present. The diallels would contain the same genotypes since the material was selected for 1 cycle. Thus, before any conclusive evidence can be offered on the inheritance of alfalfa sickness resistance or susceptibility, it will be necessary to evaluate progeny from the third cycle of selection to expose the full array of genotypes.

The method of selection used to develop resistance and susceptibility to alfalfa sickness relied on horizontal, or field resistance or susceptibility. Based on the heritability estimates (Tables 14 and 26), the selection of individual plant phenotypes was reliable. Well-replicated progeny tests minimized environmental variation in the growth room and led to the success of phenotypic recurrent selection. One disadvantage of using this form of mass selection is that in interpollinating all plants, there may be some self-fertilized seed produced. However, to overcome this problem, controlled pollination could be utilized in the form of a diallel although this places a restriction on the number of genotypes one can evaluate. Polycrossing within the populations allowed maximum expression of heterozygosity and random mating over 2 cycles of selection. This prevented inbreeding depression and utilized heterosis. As well, the number of plants selected per cycle was more than 75 which Hill *et al.* (1969) considered a minimum to suppress any appreciable change in agronomic traits not selected. Thus recurrent selection for phenotypic characters has been an effective method of allowing a wealth of genetic recombination to occur providing new source material for the next cycle of selection.

A hypothesis was put forward to explain the basis of resistance and susceptibility to alfalfa sickness. The assumption made in this theory was that alfalfa sickness resistance and susceptibility was controlled by many genes. On the basis of this premise it was thought the selection criteria of height and disease rating represented net assimilation rate (NAR) of the plant. Selecting tall plants with low disease ratings would mean the plants' capacity to manufacture assimilates would exceed the requirements of the bacteria, *Rhizobium meloloti*, and the parasitic alfalfa sickness pathogen. This is supported by Tan's (1977) work in which two resistant genotypes were found to be high yielders of dry matter and nitrogen. The remaining photosynthates would be metabolized or stored by the plant and result in high dry matter productivity. Thus a high NAR would allow the plant to live with the two organisms, and at the same time yield high returns of forage. In the susceptible genotypes, short plants with high disease ratings would have a low net liable pool of assimilates. Consequently, there would be adequate amounts of photosynthates to meet the needs of the two organisms, but little residual would be left for high dry matter yields.

To increase NAR in the alfalfa plant involves breeding for a trait inherited in a complex way. This could explain why resistance and susceptibility to alfalfa sickness are thought to be controlled by many genes and it may be the reason for the low narrow sense heritabilities obtained in the diallel strain test for disease rating (Table 14). Various components of NAR which would increase yield include larger photosynthetic area (increasing number, weight, area, or structure of leaves), fewer stems (decreasing number, length, or structure), or increasing efficiency of photosynthesis (Carlson et al., 1970). It would be interesting to compare net assimilation rates of plants from resistant genotypes, susceptible genotypes, and cultivars.

In conclusion, the overall objectives have been achieved by investigating and testing a number of physical and chemical treatments and by undertaking a plant breeding program. Heat offers the best control of the alfalfa sickness pathogen, but it is impractical. Chemical control of the pathogen using Dowco 269, would seem to be a feasible method if that chemical or a similar one were available, and commercial application were economical. Genotypes with resistance have yield advantages of 40% to 50% over unselected material and susceptible genotypes. Thus, the outlook for plant breeding seems optimistic and the ultimate control of alfalfa sickness lies in developing a cultivar with resistance to the disease.

SUMMARY

1. The agent which incites alfalfa sickness is a pythiaceous fungus. The symptoms it causes on alfalfa plants resemble those caused by _P. megasperma_ . However, the environmental conditions necessary for the survival of _P. megasperma_ are different from those associated with alfalfa sickness. Alfalfa sickness occurs on light textured soils that appear to be well-drained while _P. megasperma_ has been found in heavy soils that were poorly drained.

2. A relationship between disease rating and height, and disease rating and yield accounted for the shorter plants with lower yields when the plants were grown in sick soil. This agreed with observations that infected fields have good stands but are low yielding.

3. The alfalfa sickness pathogen attacks the root tips, noncambial lateral roots, and nodules resulting in browning, lesions, and girdling of the roots. This eventually leads to collapse and destruction of the root tissue which affects height and yield of infected plants.

4. Heat treatment of sick soil was a superior control method but it would be impractical to use on a field scale.

5. Lime, fertilizer, and moisture, at levels for optimum growth, were eliminated as caustive agents of alfalfa sickness and did not improve alfalfa growth in sick soil.

6. Height, disease rating, and yield of plants from sick soil treated with Dexon were not significantly different from plants grown in nonpasteurized sick soil, and

consequently, Dexon did not control alfalfa sickness.

7. Chemicals such as Metazoloxon and Dowco 269 were effective in controlling the alfalfa sickness pathogen. But, Dowco 269 was the more impressive and application of that fungicide as either a soil drench or soil mix satisfactorily protected alfalfa roots against the sickness pathogen.

8. High broad sense heritabilities justified the conclusion that genotypic variability exists between and within locally adapted cultivars for alfalfa sickness selection.

9. Low narrow sense heritabilities for height and disease rating in the resistant and susceptible genotypes indicated that additive genetic variation was small and successive cycles of recurrent selection would be necessary to develop high levels of resistance and low levels of susceptibility to alfalfa sickness.

10. In the field tests, results from the second year were more reliable and useful for selection purposes than those observed in the establishment year.

11. Selection progress has been slow in the two cycles of selection evaluated which implies many genes are influencing disease resistance and susceptibility.

12. Simple correlation coefficients, among height, yield, and disease rating in the growth room, were highly significant. However, correlation coefficients for agronomic characters between field and growth room were low and nonsignificant. Consequently, it is necessary to use field tests to determine the effectiveness of growth room

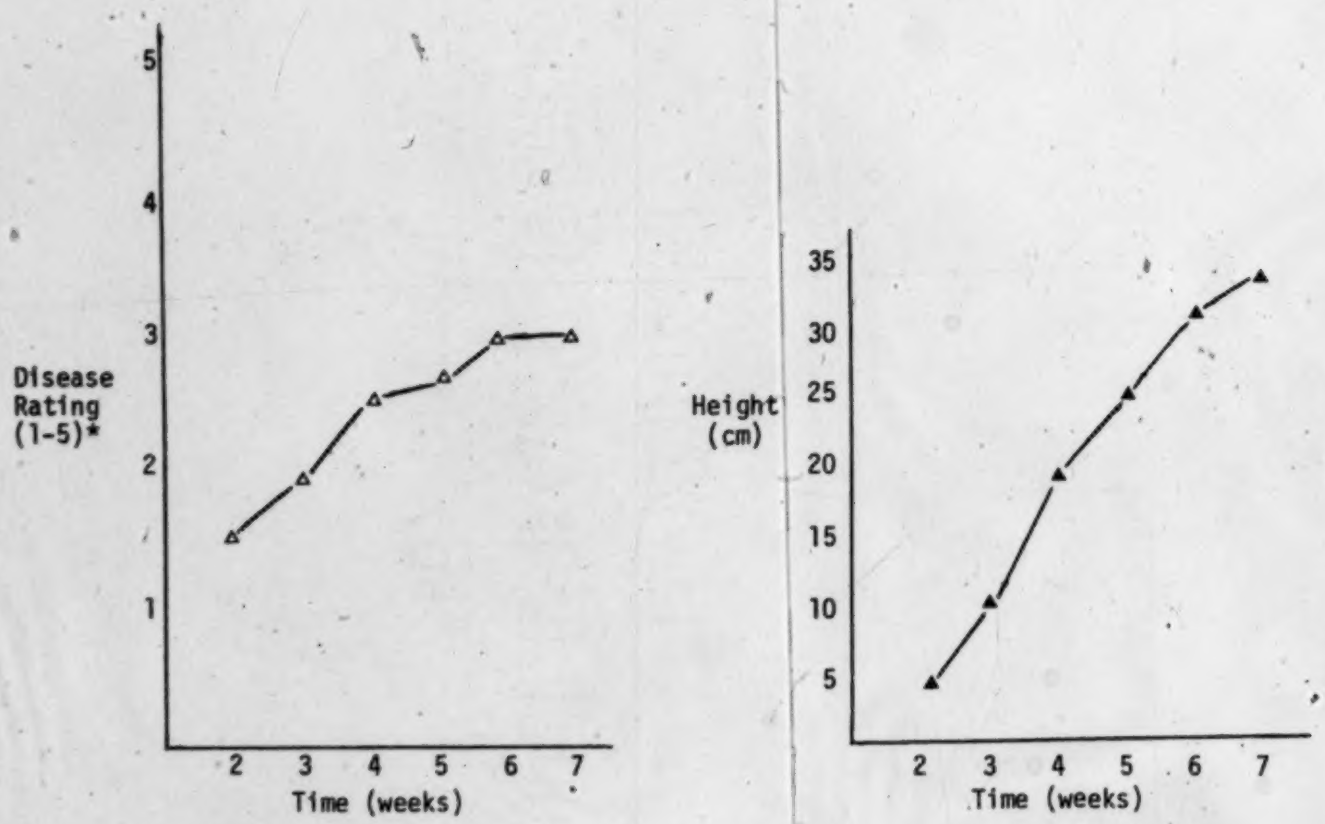
selection.

13. Recurrent phenotypic selection in the growth room was effective since high broad sense heritabilities were obtained in resistant and susceptible genotypes and there was progress in selection after 2 cycles. No major shift occurred in any of the agronomic characters not selected in the growth room according to field evaluation of the genotypes.

14. High specific combining ability variance in the resistant and susceptible diallels indicated nonadditive genetic variation was large and this was expected since the parents of the progeny had been selected for one cycle. Specific combinations of these parents could be interpollinated to form suitable two-clone hybrids while multiclone synthetics could be formulated from this material as well.

15. It was concluded that physical, chemical, and plant breeding methods of control of alfalfa sickness were discovered in this study. Pasteurization has practical limitations while fungicidal control could be expensive and short-term. Plant breeding provides a practical long-term solution to alfalfa sickness.

FIGURE 1 Height and disease rating means of Beaver seedlings for the time of infection test.



*1 = clean roots, 5 = dead plant

FIGURE 2 Frequency distributions of alfalfa seedlings in cycle 0 planted in sick soil and nonsick soil and grown in the growth room.

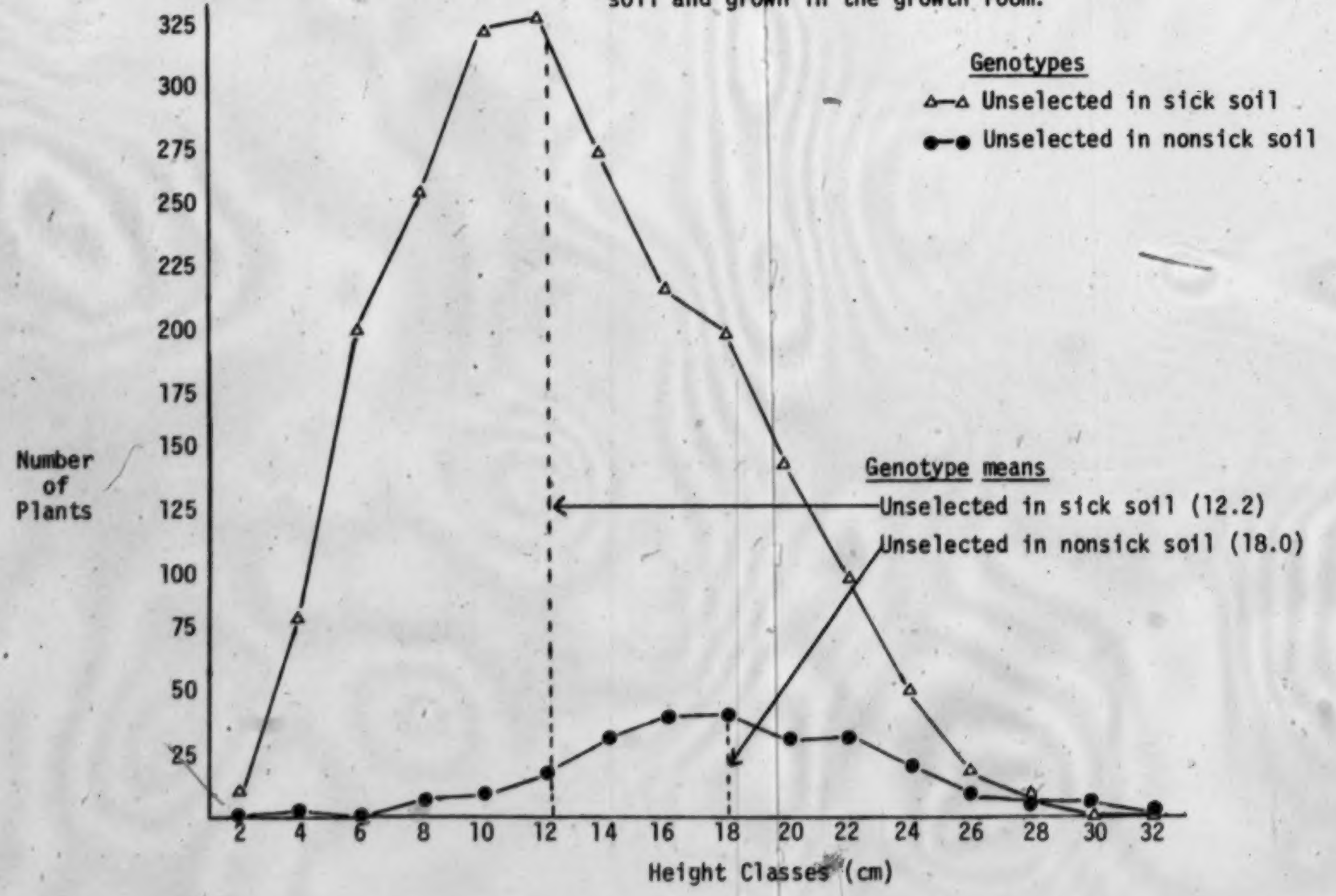


FIGURE 3 Frequency distributions of alfalfa seedling progenies in cycle 1 grown in sick soil in the growth room.

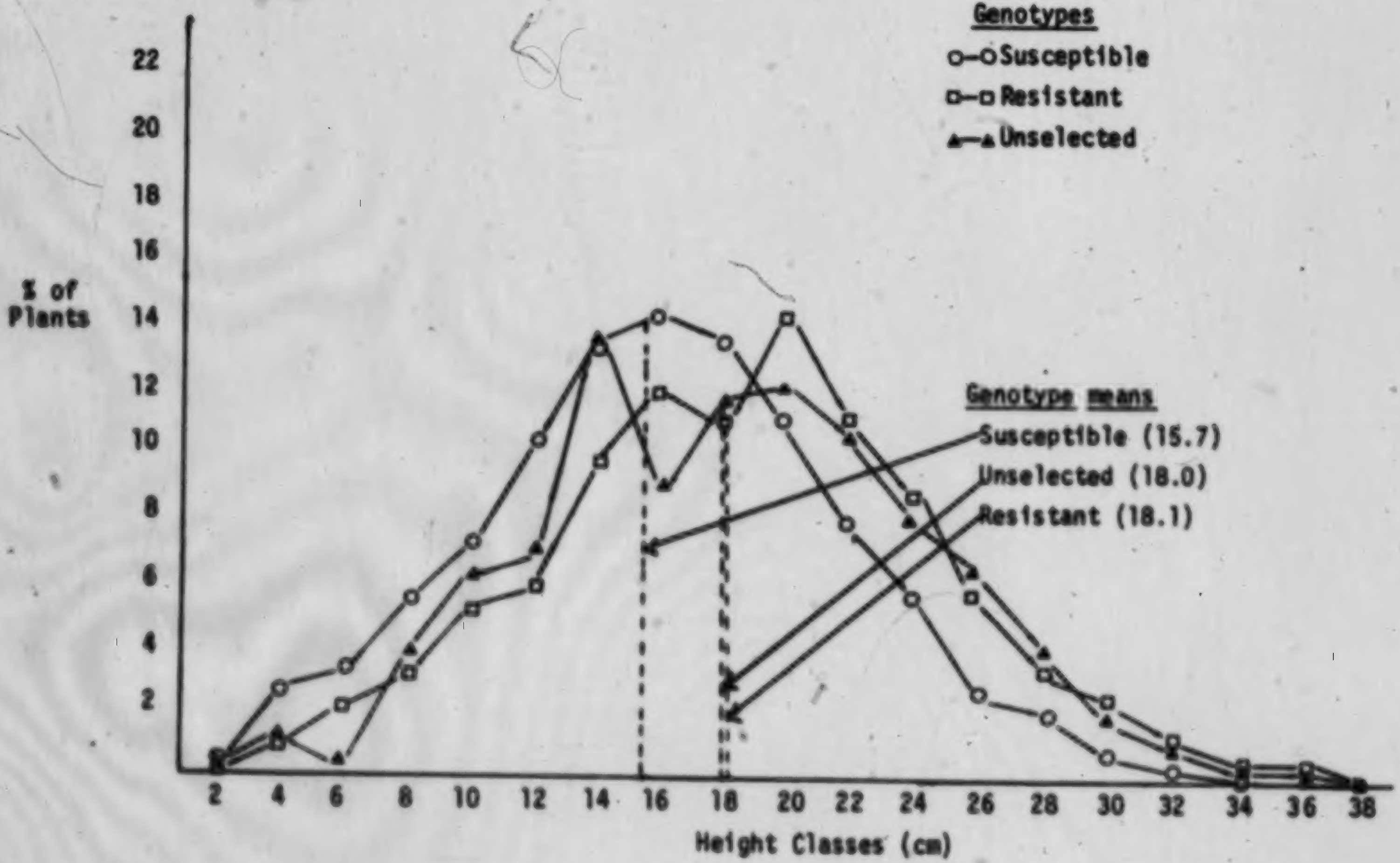


FIGURE 4 Frequency distributions of alfalfa seedling progenies in cycle 2 grown in sick soil in the growth room.

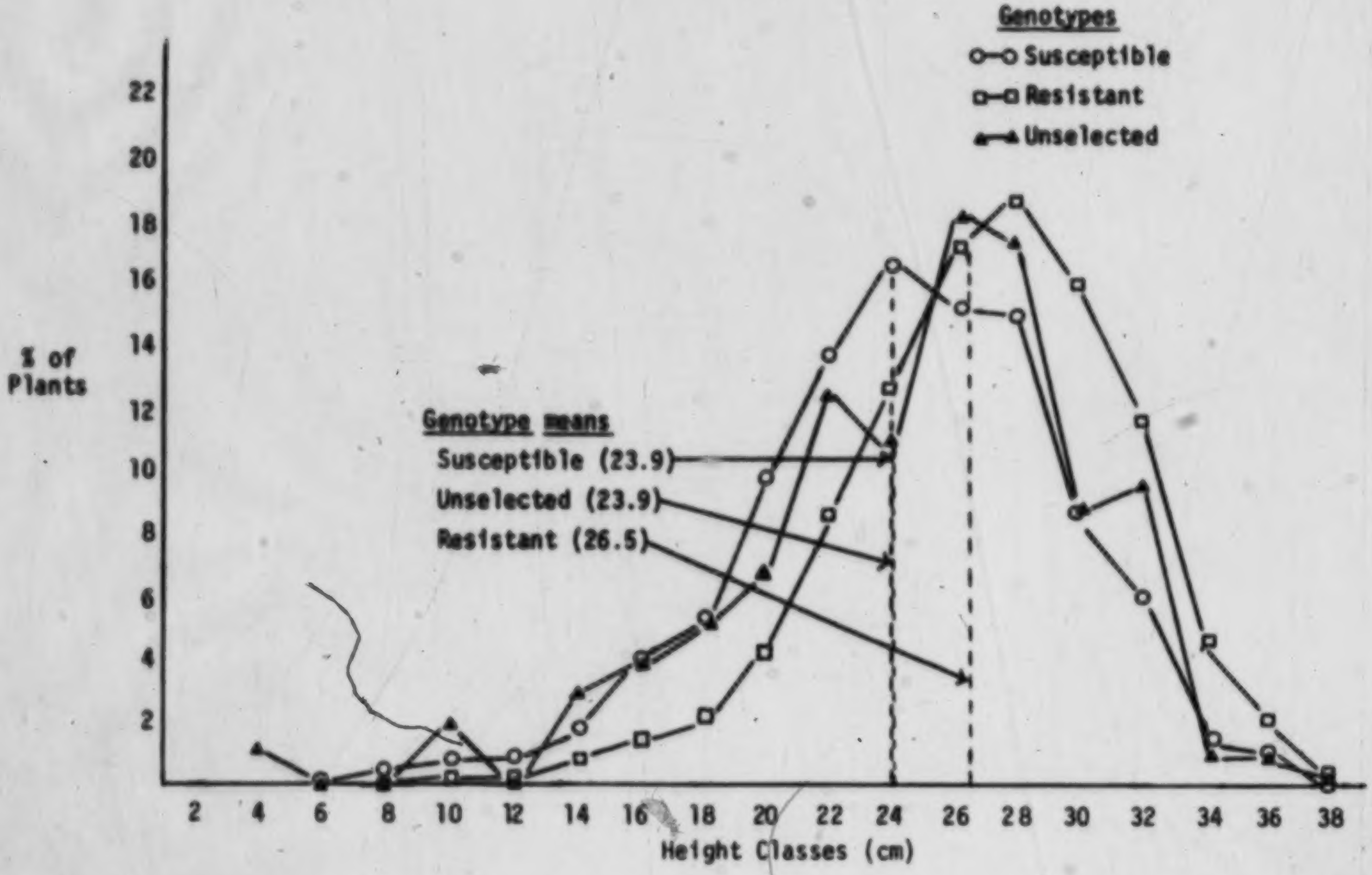


TABLE 1 Comparative morphological characteristics of Phytophthora megasperma, Phytophthora oryptogea, Pythium ultimum, and Pythium debaryanum.

Fungus	Oogonium		Oospore		Sporangium		Antherdia
	Range (μ)	Average (μ)	Range (μ)	Average (μ)	Range (μ)	Average (μ)	
Phytophthora cryptogea*	24-34	28.3	19-29	23.7	25-49 x 16-29	36.7 x 21.9	P†
Phytophthora megasperma**	42-52	47.4	37-47	41.4	15-60 x 6-45		P†
Pythium debaryanum***	15-28	21.0	12-20	17.0	15-26	19	NP†
Pythium ultimum***	19.6-22.9	20.6	14.7-18.3	16.3	12-28	20	NP†

*From Ashby, S.F. 1929. Further note on the production of sexual organs in paired cultures of species and strains of Phytophthora. Brist. Mycol. Soc. Trans. 14:254-260

**From Waterhouse, G.M. 1956. The genus Phytophthora--diagnosis and figures from original papers. Commonwealth Mycol. Inst. Mycol. Paper No. 12

***From Middleton, J.T. 1943. The taxonomy, host range, and geographic distribution of the genus Pythium. Memoirs of the Torrey Bot. Club No. 20

†P = paragynous, NP = non-paragynous

TABLE 2 A summary of the location, replication, and design of tests undertaken during 1976 and 1977.

	Test Location	Replicates per Test	Experimental Design
Pathogen Tests			
Soil test 1	growth room	3	split plot
Soil test 2	growth room	5	randomized complete block
U.S. soil test	growth room	-	observational
Time of infection test	growth room	5	randomized complete block
Inoculation test	growth room	5	randomized complete block
Fungicide test 1	growth room	5	randomized complete block
Fungicide test 2	growth room	5	randomized complete block
Strain + fungicide test	growth room	6	randomized complete block
Plant Tests			
Second cycle strain test	growth room	2	randomized complete block
Three cycle strain test	growth room	6	randomized complete block
Diallel strain test	growth room	3	randomized complete block
Lattice strain test 1	field site	4	10x10 simple lattice
Lattice strain test 2	field site	4	9x8 rectangular lattice
Strain test 1	field site	2	11x11 simple lattice
Strain test 2	field site	4	randomized complete block
Observational Strain test	field site	-	observational

TABLE 3 Height, yield, and stand survival subplot means of Beaver seedlings in soil test 1.

Treatment	Height (cm)	Yield (g)	Stand Survival (plants per pot)
Pasteurized soil	29.1 a*	3.2 a	12.7
Pasteurized-limed soil	28.1 ab	3.0 b	12.0
Nonpasteurized soil	27.0 bc	2.4 c	12.6
Nonpasteurized-limed soil	26.2 c	2.1 d	12.5
Mean	27.6	2.7	12.5
CV**(%)	9.3	22.1	13.4

*Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

**CV = coefficient of variability

TABLE 4 Height, yield, and disease rating means of Beaver seedlings in soil test 2.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Sick soil 1	20.6 b***	0.5 b	2.81 c
Sick soil 2	20.2 b	0.4 b	2.86 c
Sick soil 3	19.6 b	0.4 b	2.62 bc
Sick soil 4	19.5 b	0.4 b	2.82 c
Sick soil 5	17.3 b	0.4 b	2.57 b
Pasteurized sick soil	24.8 a	1.7 a	1.00 a
Mean	20.3	0.6	2.45
CV**(%)	4.7	25.1	6.8

*1 = clean roots, 5 = dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

TABLE 5 Height, yield, and disease rating of observation pots of Beaver seedlings in the U.S. soil test.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Minnesota soil**	27.4	3.7	3.17
Minnesota soil**	24.3	2.7	3.06
California soil**	21.7	2.4	3.00
Michigan soil**	20.1	2.4	3.44
Ontario soil**	15.6	1.7	3.79
Mean	21.8	2.6	3.29
Alfalfa sick soil***	21.2	2.3	2.52

*1 = clean roots, 5 = dead plant

**Soils reputed to be infected with _Phytophthora_ _megasperma_ were imported and tested at the Edmonton Research Station (Parkland Farm), Edmonton, Alberta, Canada

***Means are average of 5 replicates

TABLE 6 Means of height and disease rating for Beaver seedlings in the time of infection test.

Treatment	Height (cm)	Disease Rating (1-5)*
2 weeks	4.8 f***	1.54 a
3 weeks	10.2 e	1.99 b
4 weeks	20.2 d	2.55 c
5 weeks	24.6 c	2.85 d
6 weeks	30.6 b	2.98 d
7 weeks	33.4 a	2.99 d
Mean	20.6	2.47
CV**(%)	6.0	6.0

*1 = clean roots, 5 - dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

TABLE 7 Height, yield, and disease rating means of alfalfa seedling progenies in the inoculation test.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Pasteurized sick soil			
Beaver	24.3	3.5	1.00 a***
1GP130	24.0	3.0	1.00 a
1V12	24.0	2.9	1.00 a
2V96	22.8	3.3	1.00 a
1RP188	22.6	3.3	1.00 a
Pasteurized inoculated sick soil			
2V96	24.6	4.2	2.00 b
1V12	24.2	3.4	2.00 b
1GP130	23.3	3.5	2.00 b
1RP188	22.3	3.5	2.00 b
Beaver	22.2	3.2	2.00 b
Mean	23.2	3.4	1.50
Standard Deviation	1.8	0.6	0.5
CV**(%)	7.5	17.8	0.0

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*1 = clean roots, 5 = dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

TABLE 8 Mean heights, yields, and disease ratings of Beaver seedlings in fungicide test 1.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Pasteurized sick soil	32.3 a***	1.5 a	1.00 a
Non-pasteurized sick soil	26.7 b	0.7 b	2.94 b
Benlate	26.2 b	0.7 b	2.88 b
Dexon	25.4 b	0.6 b	2.86 b
Mean	27.7	0.9	2.42
CV**(%)	8.5	21.0	4.9

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*1 = clean roots, 5 = dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at the $p \leq 0.05$ by Duncan's new multiple range test

TABLE 9 Height, yield, and disease rating means of Beaver seedlings in fungicide test 2.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Pasteurized sick soil	27.8 a***	9.1 a	1.00 a
Non-pasteurized sick soil	24.2 b	4.1 cd	2.81 d
Dowco 269 Spray	24.2 b	3.9 cd	2.17 b
Dowco 269 mix	24.2 b	5.5 b	1.03 a
Metazolozon mix	23.3 b	3.2 d	2.55 c
Dowco 269 spray drench	22.7 b	5.1 bc	1.04 a
Metazoloxon mix drench	22.4 b	3.0 d	2.62 c
Dexon mix	8.3 c	0.7 e	4.00 e
Dexon mix drench	6.2 c	0.5 e	4.00 e
Mean	20.4	3.9	2.36
CV**(%)	9.1	23.6	4.6

*1 = clean roots, 5 = dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

TABLE 10 Comparison of mean heights, yields, and disease ratings of alfalfa seedling progenies in the strain + fungicide test.

Treatment	Height (cm)	Yield (g)	Disease Rating (1-5)*
Pasteurized sick soil			
1GP130	24.6 a***	3.1 ab	1.00 a
Beaver	24.5 ab	3.4 a	1.00 a
1V12	23.8 abc	2.9 abc	1.00 a
Dowco 269-sick soil			
1V12	22.7 abcde	2.6 bcd	1.00 a
Beaver	21.5 cdef	2.6 bcd	1.00 a
1GP130	18.8 gh	1.8 g	1.00 a
Non-pasteurized sick soil			
1V12	23.7 abcd	2.3 cdef	2.29 b
Beaver	20.6 efg	2.0 fg	2.99 c
1GP130	18.6 gh	1.7 g	2.97 c
Mean	22.1	2.5	1.58
CV**(%)	9.3	17.7	8.9

*1 = clean roots, 5 = dead plant
**CV = coefficient of variability
***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

TABLE 11 Means for height, yield, and disease rating of alfalfa seedling progenies in the second cycle strain test.

Genotype	Height (cm)		Yield (g)		Disease Rating (1-5)*	
	Mean	Range	Mean	Range	Mean	Range
Resistant	26.5	22.0-29.8	2.6	1.5-4.2	2.54	1.84-3.16
Susceptible	23.9	18.0-28.3	2.2	1.3-3.1	2.82	2.54-2.96
Beaver (Pasteurized sick soil)***	28.7	27.5-30.2	4.3	3.9-4.8	1.00	1.00-1.00
Beaver***	23.9	22.4-26.1	2.4	2.1-3.0	2.48	1.92-2.84
Agate****	25.7		2.6		2.85	
Mean	25.8		2.5		2.53	
Standard Deviation	2.7		0.7		0.44	
CV**(%)	6.8		15.0		6.5	

*1 = clean roots, 5 = dead plant
**CV = coefficient of variability
***Means averaged over 8 replicates
****Cultivar not included in analysis of variance

TABLE 12 Summary of mean heights, yields, and disease ratings of alfalfa seedling progenies in the three cycle strain test.

Genotype	Height (cm)	Yield (g)	Disease Rating (1-5)*
Resistant			
1V12	27.5 a***	3.5 b	2.32 bc
1V102	20.8 efg	2.0 ef	2.58 d→h
1V49	19.9 fgh	2.5 de	2.70 ghij
1V100	16.8 ij	1.2 g	2.69 f→j
Mean	21.3	2.3	2.57
2V102	26.1 ab	3.0 bcd	2.28 b
2V12	24.3 bc	3.2 bc	2.45 b→f
2V100	23.3 cde	2.6 cde	2.44 bcde
2V96	21.6 def	2.9 bcd	2.37 bcd
2V49	21.2 def	2.9 bcd	2.47 b→g
Mean	23.3	3.0	2.41
3V12B	23.5 cd	3.4 b	2.32 bc
3V96B	23.5 cd	3.0 bcd	2.32 bc
3V100	22.1 c→f	2.5 de	2.54 c→h
3V49B	21.2 c→f	2.6 cde	2.54 c→h
3V102B	20.2 fgh	1.9 ef	2.65 e→i
Mean	22.2	27	2.48
Mean(all resistant genotypes)	22.3	2.7	2.48
Susceptible			
1BP75	18.3 ghi	1.9 ef	2.63 efgh
1RP188	17.8 hij	1.9 ef	2.87 ijk
1RP159	16.8 ij	1.5 efg	2.92 jk
1GP130	16.0 ijk	1.3 efg	3.01 k
Mean	17.3	1.7	2.86
2GP130	14.0 k	1.3 efg	2.98 k
3BP75A	15.4 jk	1.2 g	2.74 jk
3GP130	14.0 k	1.2 g	2.92 hij
Mean	14.7	1.2 g	2.83
Mean(all susceptible genotypes)	16.0	1.5	2.87
Beaver (pasteurized sick soil)	28.4 a	6.0 a	1.00 a
Mean	20.6	2.4	2.53
CV**(%)	9.7	21.4	7.2

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*1 = clean roots, 5 = dead plant

**CV = coefficient of variability

***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test. When a mean is followed by more than four letters, only the beginning and last letters are written.

TABLE 13 Means of height, yield, and disease rating of alfalfa seedling progenies in the diallel strain test.

Genotype	Height (cm)	Yield (g)	Disease Rating (1-5)*
Resistant Diallel			
1V12 x 1V57†	27.0 b→f**	3.3 ef	2.32 cde
1V12 x 1B179	30.0 abc	4.1 bcde	2.21 cd
1V12 x 1B152	27.3 bcde	3.6 de	2.15 c
1V12 x 1V9	26.8 b→f	3.9 bcde	2.26 cde
1V12 x 1B103	27.8 a→e	3.7 cde	2.32 cde
1V57 x 1B179	28.2 abcd	4.0 bcde	2.34 cde
1V57 x 1B152	21.8 hijk	1.9 ghi	3.12 g
1V57 x 1V9	26.3 c→g	3.5 de	2.31 cde
1V57 x 1B103	22.6 ghij	2.2 gh	2.46 e
1B179 x 1B152	27.4 bcde	3.6 de	2.41 de
1B179 x 1V9	30.4 ab	4.2 bcd	2.23 cd
1B179 x 1B103	29.5 abc	4.4 abcd	2.23 cd
1B152 x 1V9	31.6 a	4.6 ab	2.22 cd
1B152 x 1B103	27.6 bcde	4.2 bcde	2.24 cd
1V9 x 1B103	28.1 abcd	4.7 ab	2.13 c
Mean	27.5	3.7	2.34
Susceptible Diallel			
1GP130 x 1BP113	19.2 jkl	1.7 ghi	3.06 fg
1GP130 x 1VP17	21.9 hijk	2.3 gh	2.87 f
1GP130 x 1V58	19.1 jkl	1.7 ghi	3.18 f
1GP130 x 1VP62	20.1 ijkl	2.0 ghi	3.44 h
1BP113 x 1VP17	22.6 ghij	2.1 ghi	3.18 f
1BP113 x 1VP58	20.6 h→l	1.7 ghi	3.19 f
1BP113 x 1VP62	23.3 e→l	2.2 gh	3.03 fg
1VP17 x 1VP58	17.4 l	1.2 i	3.39 h
1VP17 x 1VP62	18.2 kl	1.5 hi	3.06 fg
1VP58 x 1VP62	17.8 l	1.5 hi	3.17 g
Mean	20.0	1.8	3.15
Beaver (pasteurized sick soil)	28.7 abc	5.2 a	1.00 a
Beaver (pasteurized sick soil)	28.0 a→e	5.1 a	1.00 a
Beaver	24.0 e→l	2.5 fg	3.09 g
2V96	24.1 d→h	3.6 de	1.81 b
3V102C	24.3 d→h	3.5 de	2.20 cd
1GP130	20.9 h→l	1.9 ghi	3.04 fg
Mean	24.6	3.1	2.54
CV**(%)	8.4	15.5	4.3

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*1 = clean roots, 5 = dead plant; **CV = coefficient of variability;
***Means followed by the same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test. When a mean is followed by more than four letters only the beginning and last letters are written;
†First parent is female parent

TABLE 14 General combining ability (GCA) and specific combining ability (SCA) mean squares and F-values, ratio of GCA:SCA, heritability estimates, and genetic coefficient of variability for height and disease rating of alfalfa seedling progenies in the diallel strain test.

Genotype	Source of Variation	df	Height Mean Square	Height F	Disease Rating Mean Square	Disease Rating F
Resistant Diallel						
	Crosses	14	338.3	3.8*	2.84	22.0**
	Within crosses					
	GCA	5	13.4	27.7**	0.092	19.2**
	SCA	9	4.2	8.7**	0.047	9.9**
	GCA:SCA ratio		3:1		2:1	
	Broad sense heritability (%)		98		98	
	Narrow sense heritability (%)		46		33	
	Genetic CV†(%)		19		22	
Susceptible Diallel						
	Crosses	9	219.5	4.1**	0.92	4.9**
	Within crosses					
	GCA	4	5.8	17.0**	0.013	3.1**
	SCA	5	4.1	12.0**	0.026	6.4**
	GCA:SCA ratio		1:1		1:2	
	Broad sense heritability (%)		99		96	
	Narrow sense heritability (%)		32		11	
	Genetic CV†(%)		23		10	

**Indicates significance at the $p \le 0.01$ level; †CV = coefficient of variability

TABLE 15 Estimates of general combining ability (GCA) effects and specific combining ability (SCA) effects for height and disease rating of alfalfa seedling progenies in the diallel strain test.

Genotype			Estimates of SCA Effects						Estimates of GCA effects
Resistant diallel	Height	Parents	1V57	1B179	1B152	1V9	1B103		
		1V12	2.277	-0.251	0.027	-2.523	0.471		0.224
		1V57		1.632	-2.512	0.360	-1.757		-2.815
		1B179			-1.751	-0.190	0.560		2.380
		1B152				2.932	1.304		-0.654
		1V9					-0.579		1.519
		1B103							-0.654
								0.493 $s_{\bar{x}}^{*}$	
	Disease Rating	1V12	-0.162	0.043	-0.207	0.154	0.171		-0.091
		1V57		-0.134	0.460	-0.157	0.007		0.243
		1B179			0.021	0.071	0.001		-0.074
		1B152				-0.090	-0.184		0.132
		1V9					0.021		-0.141
		1B103							-0.069
								0.049 $s_{\bar{x}}$	
Susceptible Diallel		Parents	1BP113	1VP17	1VP58	1VP62			
	Height	1GP130	-2.885	1.944	0.841	0.100			0.364
		1BP113		0.782	0.344	-1.759			1.972
		1VP17			-1.026	-1.700			-0.258
		1VP58				-0.159			1.910
		1VP62							-0.169
								0.479 $s_{\bar{x}}$	
	Disease Rating	1GP130	0.011	-0.204	0.004	0.189			-0.043
		1BP113		0.115	-0.078	-0.048			-0.050
		1VP17			0.152	-0.063			-0.013
		1VP58				-0.078			0.113
		1VP62							-0.006
								0.053 $s_{\bar{x}}$	

*$s_{\bar{x}}$ = standard error of the means

TABLE 16 Summary of height, yield, and disease rating means of alfalfa seedling progenies in the second cycle strain test, three cycle strain test, and diallel strain test.

Genotype	Height (cm)	% of Beaver	Yield (g)	% of Beaver	Disease Rating (1-5)*	% of Beaver
Resistant	25.3	109	2.8	119	2.48	92
Susceptible	20.1	87	1.8	78	2.92	108
Beaver (pasteurized sick soil)	28.5	123	5.1	215	1.00	37
Beaver	23.1	100	2.4	100	2.71	100
Mean	23.8		2.6		2.54	

*1 = clean root, 5 = dead plant

TABLE 17 Comparison of mean heights, yields, and performance ratings of alfalfa progenies in lattice strain test 1 for 1976 and 1977.

Genotype	Height (cm)		Yield (g)		Performance Rating (5-1)*
	1976	1977	1976	1977	1976
Resistant					
Mean	51.9	72.6	294	654	3.26
Range	43.0-58.3	66.5-79.5	190-447	379-926	2.50-4.50
Susceptible					
Mean	51.7	71.8	280	606	3.20
Range	48.5-54.8	68.5-77.0	188-339	439-757	2.50-3.75
Cultivars					
Mean	48.4	70.3	258	608	2.97
Range	46.8-53.9	69.0-71.8	216-365	538-689	2.38-3.38
Mean	51.6	72.3	290	646	3.22
Standard Deviation	4.6	4.3	89	156	0.68
CV**(%)	6.9	4.8	25.5	20.0	18.8

*5 = productive stand, 1 = unproductive stand

**CV = coefficient of variability

TABLE 18 Comparison of mean heights and yields of alfalfa progenies in lattice strain test 2 for 1976 and 1977.

Genotype	Height (cm) 1976	Height (cm) 1977	Yield (g) 1977
Resistant			
Mean	44.5	67.9	323
Range	36.7-51.6	62.2-72.5	223-405
Susceptible			
Mean	45.9	67.6	327
Range	35.7-52.0	60.7-73.9	210-526
Cultivars			
Mean	45.4	68.4	348
Range	42.7-49.0	67.0-70.2	309-395
Mean	45.0	67.9	327
Standard Deviation	8.4	5.1	101
CV*(%)	10.6	5.9	27.6

*CV = coefficient of variability

TABLE 19 Mean heights and yields of alfalfa progenies in strain test 1 for 1976 and 1977.

Genotype	Height (cm) 1976	Height (cm) 1977	Yield (g) 1977
Resistant			
Mean	33.0	61.4	311
Range	24.0-41.0	53.0-72.0	140-501
Susceptible			
Mean	34.3	61.2	275
Range	26.0-43.0	55.0-68.0	155-404
Cultivars			
Mean	36.3	64.8	315
Range	29.5-39.7	60.5-72.3	278-386
Mean	33.7	61.7	301
Standard Deviation	5.2	5.2	97
CV*(%)	11.6	7.0	27

*CV = coefficient of variability

TABLE 20 Height and disease rating means of alfalfa progenies in strain test 2 for 1976.

Genotype	Height (cm)	Disease Rating (1-5)*
Resistant		
2V96	48.5 a**	2.53 a
1V12	46.0 ab	2.70 abcd
2V12	42.5 abc	2.73 abcd
1B179	40.5 abcd	2.75 abcd
1G169	40.0 bcd	2.60 ab
1R188	31.8 e	2.85 cd
Mean	41.5	2.69
Susceptible		
2BP69	44.0 abc	2.65 abc
1BP113	42.0 abcd	2.90 d
1GP33	41.0 bcd	2.75 abcd
2VP98	41.0 bcd	2.90 d
1VP17	39.0 b	2.73 abcd
1RP188	34.8 de	2.63 abc
Mean	40.3	2.76
Cultivars		
Grimm	42.0 abcd	2.83 bcd
Vernal	41.0 bcd	2.78 bcd
Beaver	38.0 cde	2.73 abcd
Roamer	37.5 de	2.75 abcd
Mean	39.6	2.77
Mean	40.6	2.74
CV(%)***	10.7	5.2

*1 = clean roots, 5 = dead plant

**Means followed by same letter are not significantly different at $p \leq 0.05$ by Duncan's new multiple range test

***CV = coefficient of variability

TABLE 21 Summary of alfalfa progeny height and yield means for each field test in 1976 and 1977.

Field Test		Year	Mean	CV*(%)	Standard Deviation	Range of Means
Lattice strain test 1	Height (cm)	1976	51.6	6.9	4.6	43.0-58.3
		1977	72.3	4.8	4.3	66.5-79.5
		1976-77**	62.0	6.4	11.3	56.4-68.0
	Yield (g)	1976	290	25.5	89	188-447
		1977	646	20	156	399-926
		1976-77	468	28	218	296-620
Lattice strain test 2	Height (cm)	1976	45.0	10.6	8.4	35.7-52.0
		1977	67.9	5.9	5.1	60.7-73.9
		1976-77	56.4	11.3	13.4	47.8-61.8
	Yield (g)	1977	327	27.6	101	210-526
Strain test 1	Height (cm)	1976	33.7	11.6	5.2	24.0-43.0
		1977	61.7	7.0	5.2	53.0-72.3
		1976-77	47.7	10.7	15.0	39.5-56.5
	Yield (g)	1977	301	27.0	97	140-501
Strain test 2	Height (cm)	1976	40.6	10.7	5.8	31.8-48.5
Observational strain test***	Height (cm)	1976	49.9	-	-	28-70
		1977	68.1	-	-	54-80
	Yield (g)	1977	462	-	-	168-951

*CV = coefficient of variability

**Represents the analysis of the agronomic character over two years

***Not replicated

TABLE 22 Simple correlation coefficients of agronomic characters for growth room tests involving the alfalfa sickness pathogen.

Growth Room Test	Height vs Yield	Height vs Disease Rating	Yield vs Disease Rating
Soil test 1	0.82**	NC	NC
Soil test 2	0.78**	-0.70**	-0.92**
Time of infection test	NC†	0.95**	NC
Inoculation test	0.43**	-0.06	0.28
Fungicide test 1	0.87**	-0.78**	-0.90**
Fungicide test 2	0.80**	-0.80**	-0.85**
Strain + fungicide test	0.84**	-0.36*	-0.52**

*,**Indicate significance at the $p \leq 0.05$ and 0.01 levels, respectively

†NC = one of the agronomic characters was not studied, hence no correlation coefficient

TABLE 23 Simple correlation coefficients of agronomic characters for growth room tests involving the alfalfa plant.

Growth Room Test	Height vs Yield	Height vs Disease Rating	Yield vs Disease Rating
Second cycle strain test	0.69**	-0.27**	-0.52**
Three cycle strain test	0.78**	-0.70**	-0.78**
Diallel strain test	0.90**	-0.71**	-0.86**

**Indicates significance at the $p \leq 0.01$ level

TABLE 24 Simple correlation coefficients of agronomic characters for field tests involving the alfalfa plant in 1976 and 1977.

Agronomic Character	Field Test	Height (1977)	Yield (1976)	Yield (1977)	Performance Rating (1976)	Disease Rating (1976)
Height (1976)	Lattice strain test 1	0.43**	0.68**	0.37**	0.71**	NC
	Lattice strain test 2	0.65**	NC	0.59**	NC	NC
	Strain test 1	0.59**	NC	0.44**	NC	NC
	Strain test 2	NC†	NC	NC	NC	0.19
Height (1977)	Lattice strain test 1		0.33**	0.53**	0.46**	NC
	Lattice strain test 2		NC	0.69**	NC	NC
	Strain test 1		NC	0.71**	NC	NC
Yield (1976)	Lattice strain test 1			0.58**	0.66**	NC
Yield (1977)	Lattice strain test 1				0.49**	NC
	Lattice strain test 2				NC	NC
	Strain test 1				NC	NC
Performance Rating (1976)	Lattice strain test 1					NC
Disease Rating (1976)	Strain test 2					

**Indicates significance at $p \leq 0.01$ level

†NC = one of the agronomic characters was not studied, hence no correlation coefficient

TABLE 25 Mean heights and disease ratings, percent of susceptible genotypes, and selection intensity of alfalfa seedling progenies derived from 2 cycles of selection for resistance and susceptibility to alfalfa sickness in the growth room.

Cycle	Genotype	Height	% of Susceptible	Disease Rating	% of Susceptible	Selection Intensity for Height and Disease Rating
0	Unselected	12.2	-	1.41	-	13
1	Susceptible	15.7	100	1.27	100	7
	Resistant	18.1	115	1.30	102	6
2	Susceptible	23.9	100	2.82	100	6
	Resistant	26.5	110	2.54	90	8

TABLE 26 Means, regression coefficients (b), and narrow sense heritabilities of height and disease rating for alfalfa seedling progenies selected in response to alfalfa sickness over two cycles in the growth room.

Cycle	Genotype	Height (cm)	b	Narrow Sense Heritability (%)	Disease Rating (1-5)†	b	Narrow Sense Heritability (%)
0	Unselected parents	14.7	-	-	1.34††	-	-
1	Resistant + susceptible	17.0	0.15**	30	1.29††	0.06*	12
1	Susceptible	15.7	0.02		1.27††	0.06*	12
1	Resistant	18.1	0.11		1.30††	0.07	
1	Cycle one parents	23.1	-	-	1.29††	-	-
2	Resistant + susceptible	25.8	0.09**	18	2.62	0.11*	22
2	Susceptible	23.9	-0.11		2.83	0.09**	18
2	Resistant	26.5	-0.03		2.54	0.07	

*,**Indicates significance at $p \leq 0.05$ and 0.01 levels, respectively
†1 = clean roots, 5 = dead plant
††1 = clean roots, 4 = dead plant

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APPENDIX 1 Soil characteristics of samples taken from the area selected for the field site*, 1976.

Sample Number	Sample Area	Sample Depth (cm)	pH	Available Nutrients (lb/acre) N	P	K	Soil Conductivity (mmhos/cm)
1	Lattice strain test 1	0-15	6.0	11	48	147	0.1
2	Lattice strain test 1	15-30	6.2	13	28	107	0.1
3	Lattice strain test 1	0-15	6.0	21	66	251	0.2
4	Lattice strain test 1	15-30	6.2	22	37	136	0.2
5	Lattice strain test 2	0-15	6.0	25	108	313	0.2
6	Lattice strain test 2	15-30	6.0	13	74	160	0.1
7	Strain test 1	0-15	5.9	22	111	203	0.2
8	Strain test 1	15-30	6.5	13	74	130	0.1
9	J**	0-15	5.9	44	82	235	0.2
10	J**	15-30	6.3	12	49	106	0.1
11	J**	0-15	6.0	7	33	145	0.1
12	J**	0-15	6.0	11	25	179	0.1

*From Soil and Feed Testing Laboratory, O.S. Longman Bldg., Alberta Agriculture, Edmonton, Alberta

**Samples taken in field adjacent to field plot

APPENDIX 2 Weather data for the field site, Spruce Grove, Alberta, from 1967 to 1976*.

Year	Annual Precipitation (cm)	Rainfall** (cm)	Mean Daily Temperature** (°C)	Daily Temperature (°C)**	
				Maximum	Minimum
1967	34.9	15.3	14.6	20.4	8.7
1968	40.8	22.6	13.4	19.2	7.7
1969	50.0	28.7	14.4	20.3	8.4
1970	48.2	29.4	15.2	20.8	9.4
1971	57.1	34.7	14.9	20.6	9.2
1972	51.9	29.4	14.7	20.0	9.3
1973	63.6	40.9	14.1	19.8	8.3
1974	52.9	30.7	13.3	17.8	7.9
1975	60.2	43.1	13.7	19.0	8.4
1976	50.0	32.5	14.4	19.9	9.1
Mean	49.8	30.5	14.8	19.8	8.8

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*From Environment Canada. 1967-1977. Annual meteorological summary for Spruce Grove, Alberta. Environment Service, Environment Canada, Edmonton, Alberta, Canada

**For the months May to August inclusive

APPENDIX 3 Comparison of mean heights, yields, and disease ratings of alfalfa seedling progenies in the second cycle strain test.

Genotype	Height (cm)	Yield (g)	Disease Rating (1-5)*
Resistant			
2B180	29.8	2.6	2.04
2R187	26.7	2.7	2.79
2V18	29.6	3.1	2.46
2B179	29.6	2.9	2.42
2B183	29.6	2.8	2.31
2V102	29.2	3.1	2.31
2B76	28.8	2.7	2.73
2V15	28.7	3.2	2.84
2V96	28.3	4.2	2.30
2B178	28.2	2.5	2.62
2V11	28.1	2.6	2.57
2V58	28.0	2.8	2.46
2V59	28.0	2.7	2.38
2G173	27.8	3.0	2.72
2V8	27.7	2.9	2.42
2B75	27.7	2.7	2.69
2V45	27.7	2.6	2.50
2R1	27.5	2.7	2.77
2V13	27.4	3.0	2.54
2V62	27.4	2.7	2.38
2V17	27.4	2.7	2.50
2B104	27.4	2.4	2.77
2V94	27.3	2.9	2.46
2V10	27.3	2.8	2.77
2V12	27.2	3.7	2.19
2G130	27.2	2.3	2.54
2V43	27.2	2.2	2.77
2V14	27.0	2.5	2.56
2V51	27.0	2.3	2.38
2V49	26.9	3.0	2.27
2B72	26.8	2.6	3.16
2B108	26.7	2.8	2.61
2R186	26.6	2.8	2.88
2G129	26.6	2.3	2.77
2V53	26.5	2.5	2.27
2V52	26.4	2.5	2.54
2B69A	26.3	2.3	2.65
2V44A	26.1	2.6	1.84
2R165	25.9	2.3	2.58
2B106A	25.9	2.3	2.80
2V46	25.7	1.9	2.34
2R86	25.6	2.4	2.54
2B73	25.6	2.2	2.16

APPENDIX 3 Continued

Genotype	Height (cm)	Yield (g)	Disease Rating (1-5)*
2G169	25.5	3.0	2.77
2B24	25.5	2.4	2.26
2V56	25.4	2.8	2.35
2B66A	25.3	2.0	2.50
2B111	24.7	2.3	2.80
2R188	24.5	1.9	2.62
2B117A	24.2	2.2	2.76
2R66	23.7	2.7	2.84
2R26	23.7	2.1	2.92
2B74	23.6	2.1	2.54
2G127	23.5	2.0	2.38
2V99	23.3	2.3	2.46
2B29	22.8	1.5	2.08
2B110	22.7	2.1	2.62
2B70	22.5	2.2	2.54
2B6	22.0	1.8	2.66
Susceptible			
2BP71	28.3	2.5	2.65
2VP102	26.8	2.6	2.96
2VP53	26.0	2.5	2.88
2GP33	25.7	3.1	2.81
2VP101	25.5	2.6	2.88
2VP48	25.0	2.7	2.54
2VP52	25.0	2.2	2.96
2BP105	24.9	1.8	2.76
2BP6	24.8	1.8	2.62
2RP147	24.6	2.7	2.92
2RP26	24.2	2.2	2.92
2BP69	24.2	2.0	2.73
2VP17	23.7	1.8	2.92
2VP43	23.6	2.2	2.92
2BP113	23.5	1.7	2.66
2VP46	23.3	2.2	2.77
2VP62	23.0	2.1	2.96
2VP61	23.0	1.8	2.96
2BP115	22.9	2.2	2.88
2GP130	22.4	2.7	2.69
2BP75	21.2	1.6	2.80
2BP153	20.7	1.6	2.74
2VP60	18.0	1.3	2.84
Cultivars			
Beaver (pasteurized soil)**	28.7	4.3	1.00

APPENDIX 3 Continued

Genotype	Height (cm)	Yield (g)	Disease Rating (1-5)*
Beaver**	24.0	2.4	2.48
Agate***	25.7	2.6	2.85
LSD (0.05)†	3.5	0.8	0.33
LSD (0.01)†	4.6	1.0	0.43
LSD (0.05)††	2.7	0.6	0.26
LSD (0.01)††	3.6	0.8	0.34

*1 = clean roots, 5 = dead plant

**Means averaged over 8 replicates

***Cultivar not included in the analysis of variance

†LSD = least significant difference between means of resistant and susceptible genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

††LSD = least significant difference between means of resistant, susceptible, and cultivar genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

APPENDIX 4 Comparison of mean heights, yields, and performance ratings of alfalfa progenies in lattice strain test 1 for 1976 and 1977.

Genotype	Height (cm)		Yield (g)		Performance Rating (5-1)*
	1976	1977	1976	1977	1976
Resistant					
2B66A	58.3	77.0	388	730	4.25
2V96	57.3	75.0	439	751	4.00
1B24	56.0	75.0	354	794	4.00
2B29	56.5	79.5	317	689	3.75
1B29	56.0	76.5	354	824	4.00
2R163	56.0	72.5	447	763	4.00
2V100	55.8	71.5	403	582	3.50
1B117	55.5	75.0	294	741	3.25
2V15	55.3	76.5	344	703	3.50
2B106	55.3	75.5	367	684	4.00
1V15	55.3	73.5	350	554	3.25
2V61A	55.0	73.0	377	608	3.75
1V11	54.5	75.5	314	631	3.50
1V57	54.5	71.0	446	602	3.50
2V19	54.5	69.5	330	517	3.25
2V48A	54.3	75.0	365	724	4.00
2V7	54.0	74.5	286	613	3.50
1B73	54.0	73.0	437	802	3.75
2B76	53.8	77.0	310	858	3.75
2R86	53.8	72.5	357	705	3.00
1B110	53.5	78.5	304	857	3.50
2V99	53.5	72.0	330	713	3.25
2B115	53.3	76.5	240	682	3.00
1V41	53.3	76.0	288	677	3.50
1V49	53.3	74.5	338	793	3.50
2B6	53.3	72.0	356	648	4.00
2V61	53.3	70.5	295	515	3.25
1V9	53.0	74.3	353	720	3.25
2V102A	53.0	70.0	305	585	3.50
2V94	52.8	77.0	247	613	3.25
2B115A	52.8	76.0	252	708	3.25
2V42	52.8	73.0	285	681	3.25
2V10	52.8	71.5	276	589	3.25
1R26	52.5	75.0	272	639	3.00
2V17	52.5	73.5	294	672	3.25
2G169	52.5	69.0	250	706	3.25
2V13	52.3	73.0	333	842	3.50
1B108	52.0	78.0	212	729	3.25
1G130	52.0	71.5	247	628	3.25
1B75	51.8	77.0	256	727	3.50

APPENDIX 4 Continued

Genotype	Height (cm)		Yield (g)		Performance Rating (5-1)*
	1976	1977	1976	1977	1976
1B109	51.8	74.5	268	674	3.50
1B69	51.8	73.5	369	627	3.25
1V12	51.5	74.5	296	718	3.00
1V17	51.5	72.0	292	536	3.00
2V60A	51.5	69.0	267	424	3.00
2V14	51.5	67.0	194	399	2.75
1G127	51.3	72.0	314	673	3.00
1B179	51.3	71.5	258	606	3.25
2V56	51.3	68.0	278	550	3.00
2B151	51.0	76.0	266	759	3.25
2B75	51.0	75.0	256	780	3.25
2B183	51.0	72.0	309	725	3.25
2V49A	51.0	71.5	329	692	3.25
1V102	51.0	71.5	298	600	3.00
1B104	51.0	68.5	252	555	3.00
2R165	50.8	69.5	285	600	3.00
2R187	50.5	76.5	305	926	3.75
2R26	50.5	73.5	241	690	3.00
2V62	50.5	72.5	310	642	3.00
1V8	50.5	72.0	226	582	3.00
1V51	50.5	66.5	205	481	2.50
1B74	50.3	72.0	237	583	3.25
2V51A	50.0	68.5	324	592	3.25
2V49	50.0	68.0	245	503	2.75
2V46	49.8	73.5	191	513	2.75
1B70	49.8	73.0	259	802	3.25
2R146	49.8	70.5	259	547	3.00
1V46	49.8	70.0	281	571	2.75
2V58	49.8	67.5	242	615	3.00
1G169	49.5	72.0	289	785	3.50
2R30	49.5	71.5	251	581	3.75
1B150	49.3	71.5	266	658	3.25
2V100A	49.3	71.5	248	551	3.00
2V48	49.0	70.0	269	578	2.75
2V59	48.8	68.0	198	538	2.75
1R147	48.5	71.5	254	635	3.00
2V12	48.3	70.0	210	446	2.50
2B23	48.0	71.0	287	628	3.00
1R165	46.5	71.0	271	802	3.00
2B113	45.0	70.5	223	620	2.50
2B70	44.3	67.0	190	553	2.50
2R148	43.0	71.5	294	678	2.50
Susceptible					
2BP113	54.8	77.0	308	692	3.75
1RP159	54.0	75.0	339	757	3.25

APPENDIX 4 Continued

Genotype	Height (cm) 1976	Height (cm) 1977	Yield (g) 1976	Yield (g) 1977	Performance Rating (5-1)* 1976
1VP62	53.0	70.5	276	627	3.25
1BP113	52.5	73.0	288	587	3.50
2BP69	52.5	70.0	318	591	3.50
1GP33	52.3	70.5	314	651	3.75
1GP130	50.5	68.5	188	450	2.75
1RP188	49.5	69.0	288	585	2.50
1VP9	49.3	72.0	281	678	2.75
1VP17	48.5	72.5	200	439	3.00
Cultivars					
Vernal**	53.9	71.8	365	689	3.38
Roamer**	48.9	69.0	230	584	2.38
Beaver**	47.3	71.0	219	623	3.13
Grimm**	46.8	69.5	216	538	3.00
LSD (0.05)†	4.9	4.9	103	179	0.80
LSD (0.01)†	6.5	6.4	135	236	1.05
LSD (0.05)††	4.3	4.2	89	155	0.70
LSD (0.01)††	5.6	5.5	117	204	0.91

*5 = productive stand, 1 = unproductive stand

**Means are average of 8 replicates

†LSD = least significant difference between means of resistant and susceptible genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

††LSD = least significant difference between means of resistant, susceptible and cultivar genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

APPENDIX 5 Height and yield means of alfalfa progenies in lattice strain test 2 for 1976 and 1977.

Genotype	Height (cm)		Yield (g)
	1976	1977	1977
Resistant			
2B183A	51.6	70.6	372
1V61	50.4	69.3	262
1V43	49.5	68.2	346
1V44	48.6	69.6	306
2V8	48.5	72.5	361
1G20	47.7	69.6	396
2V99A	47.5	68.4	360
2V44A	47.4	71.2	337
1G35	47.3	64.6	291
2V102	47.2	67.5	405
2V50	47.1	68.8	352
2V62A	47.1	68.1	372
1B182	46.8	69.0	314
1R146	46.8	67.3	330
2R137	46.2	70.2	358
2V47	46.2	69.2	319
1V45	46.2	69.0	321
1B178	46.2	68.0	363
2G129	45.6	68.3	323
2V52A	45.4	70.9	343
2B104	45.3	64.5	323
1G4	45.0	66.6	351
1G157	44.8	71.3	371
1V16	44.6	67.3	342
2G158	43.6	72.5	399
1R137	43.4	66.6	338
2V11	43.1	68.6	235
1G177	43.1	66.8	288
1B151	42.2	72.1	348
2B76A	42.2	68.0	263
1R145	42.1	66.3	251
2R88	42.0	66.6	308
2B155	41.8	66.1	292
1V18	41.7	66.5	262
1R188	41.3	68.3	324
1B181	41.3	67.2	308
2G173	41.1	62.3	319
1V59	40.1	63.0	223
1R32	39.0	69.5	357
1R25	37.8	64.8	295
1R83	37.2	71.3	256
1G37	36.7	62.2	266

APPENDIX 5 Continued

Genotype	Height (cm) 1976	Height (cm) 1977	Yield (g) 1977
Susceptible			
2BP155	52.0	72.8	377
2VP98	51.2	67.4	305
1VP100	50.6	69.9	357
1GP34	50.2	67.7	291
2BP105	50.0	73.9	526
2GP33	50.0	69.9	376
1VP17	49.2	68.7	289
2VP53	48.9	71.1	439
2VP54	48.1	65.9	210
2VP55	47.1	68.1	383
2VP96	45.9	72.4	432
2VP14	45.8	66.1	342
1VP58	45.2	64.7	272
1BP151	44.5	65.4	329
2GP130	44.2	64.5	284
2GP34	44.0	66.0	338
2VP43	43.9	66.3	268
2VP42	42.1	68.5	300
1BP70	41.1	64.8	298
1GP133	40.6	65.7	256
1GP127	39.7	66.0	292
1GP21	35.7	60.7	225
Cultivars			
Vernal*	49.0	69.3	341
Beaver*	45.3	70.2	395
Grimm*	44.6	67.2	348
Roamer*	42.7	67.0	309
LSD (0.05)†	6.6	5.6	125
LSD (0.01)†	8.7	7.3	164
LSD (0.05)††	5.7	4.8	108
LSD (0.01)††	7.6	6.4	143

*Means averaged over 8 replicates

†LSD = least significant difference between means of resistant and susceptible genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

††LSD = least significant difference between means of resistant, susceptible and cultivar genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

APPENDIX 6 Mean heights and yields of alfalfa progenies in strain test 1 for 1976 and 1977.

Genotype	Height (cm)		Yield (g)
	1976	1977	1977
Resistant			
2V56A	41	72	460
1V13	41	68	412
2V53	40	70	373
2V51	40	65	470
2V46A	40	62	325
1V93	40	61	269
1V19	39	72	360
2B111	39	66	344
2B75A	39	65	442
1V50	39	65	411
2V45	39	64	335
1V54	38	68	259
2B72	38	63	375
1V52	38	61	336
1G192	37	66	357
2V94A	37	63	337
2B110	36	69	481
2B74	36	68	451
2V101	36	63	226
2V95A	36	61	237
2B67	36	59	269
1V14	36	57	140
1R30	35	65	287
1B66	35	64	377
1R90	35	63	310
2V45A	35	60	306
1V53	35	58	245
2B108A	34	70	434
1V96	34	64	376
1V97	34	64	327
1G90	34	61	328
2B73	34	61	313
1G129	34	59	258
1B67	33	68	501
2R186	33	64	416
1G134	33	62	328
1V99	33	59	252
1G34	33	58	331
2B74A	32	68	398
1R82	32	64	317
2B185A	32	59	345
1G133	32	59	282
2R159	32	59	262

APPENDIX 6 Continued

Genotype	Height (cm)		Yield (g)
	1976	1977	1977
1B184	32	58	320
1V100	32	55	160
1G126	31	63	292
1R148	31	61	380
1B113	31	61	301
1G171	31	60	295
1G28	31	58	282
1G189	31	55	203
1R84	30	63	218
1G27	30	62	341
2B112A	30	61	314
1B111	30	59	282
1G174	30	59	255
2G193	30	54	203
1R163	29	63	301
1B72	29	62	256
1B155	29	60	263
1G131	29	57	237
1V10	29	53	147
1R88	28	64	375
1B23	28	60	308
1R144	28	59	387
1G175	28	59	315
1B153	28	57	204
1B180	28	56	169
1G173	27	58	342
1G132	27	56	224
1B68	25	59	251
1R31	25	56	171
1R139	24	58	227
1G158	24	55	237
Susceptible			
1VP47	43	68	334
1VP49	40	63	221
2BP6	39	67	348
1BP67	39	64	284
1VP99	39	62	278
2BP67	38	68	404
1GP129	38	64	345
1BP75	38	61	235
2VP49	37	66	357
1VP42	37	64	266
1VP8	37	63	288
1VP51	37	59	201

APPENDIX 6 Continued

Genotype	Height (cm) 1976	Height (cm) 1977	Yield (g) 1977
1VP60	37	55	212
2RP147	36	62	300
1VP16	36	61	220
2BP75	35	66	299
2BP71	35	64	361
1RP165	35	63	233
2VP61	35	61	325
1GP4	35	61	230
1GP28	35	56	287
1RP31	34	61	346
1BP179	33	66	356
1VP50	33	58	239
1GP192	31	60	296
1BP183	31	59	237
1GP126	30	61	308
1GP132	30	61	288
1VP19	30	55	155
1VP57	29	57	274
1VP15	29	56	256
1RP86	28	59	234
1GP20	28	58	205
1RP1	27	55	198
1RP62	26	59	214
Cultivars			
Vernal*	40	64	291
Beaver*	39	72	386
Agate*	37	61	278
Roamer*	34	64	288
Grimm*	30	61	309
LSD (0.05)†	5.5	6.0	115
LSD (0.01)†	7.3	8.0	152
LSD (0.05)††	4.8	5.2	100
LSD (0.01)††	6.3	6.9	132

*Means averaged over 4 replicates

†LSD = least significant difference between means of resistant and susceptible genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively

††LSD = least significant difference between means of resistant, susceptible and cultivar genotypes at $p \leq 0.05$ and $p \leq 0.01$ levels, respectively